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ANATOMIC AND CLINICAL RE-EVALUATION OF CAMPER'S, SCARPA'S, AND COLLES' FASCIAE

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THE subcutaneous tissue of the ventral abdominal wall, particulary that distal to the umbilicus, is described in the anatomic and clinical texts as consisting of a superficial fatty layer (Camper's fascia) and a deeper membranous layer (Scarpa's fascia). These two layers supposedly fuse and continue caudally as the dartos layer around the penis and scrotum, wherein the continuity of Camper's fascia is not fatty, and involuntary muscle fibers are imbedded in these fused layers. In the perineum, the continuations of the two layers are described as having the same pattern as that found over the ventral abdominal wall—a superficial fatty layer in the urogenital triangle, continuous with the fat in the ischiorectal fossa, and a deeper membranous layer (Colles' fascia) which is adherent to the inferior rami of the pubis and ischium and to the posterior border of the urogenital diaphragm. The continuity of the membranous layer into the ischiorectal fossa is not well established.

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Fig. 18. Kodachrome of a sagittal section through the pelvis from a male cadaver with simulated urinary extravasation from the penile urethra. The red liquid latex injected into the perforated penile urethra has infiltrated all of the subcutaneous tissue between the dermis and: the layers of Buck's fascia over the components of the penis in the perineum; the tunica vaginalis in the scrotum; Buck's fascia along the shaft of the penis; the intrinsic fascia over the aponeurosis of the external oblique muscle; and into the superficial part of the ischiorectal fossa. Note that the simulated extravasate is not limited by an inner membranous layer (Scarpa's-Colles' fasciae) but infiltrates the entire thickness of the subcutaneous tissue. ×0.17.

Fig. 19. Kodachrome of a partial dissection of the subcutaneous tissue of the lower abdominal wall, the penis, and scrotum from a male cadaver with simulated urinary extravasation from the penile urethra. The skin has been removed from the areas shown. Note the red latex, within the entire thickness of the subcutaneous tissue in the scrotum and immediately above the pubic symphysis. A larger quantity of simulated extravasate would have filled the entire thickness of the subcutaneous more cephalad over the ventral abdominal wall. ×0.25.

Fig. 20. Kodachrome of a dissection from a simulated case of extravasation. The skin has been removed and the layers of spermatic fasciae have been opened and retracted. The simulated extravasate infiltrated the layers of spermatic fasciae superficial to the tunica vaginalis testis and ascended retrogradely upward into spermatic cord. ×0.3.

Fig. 21. Kodachrome of a dissection from a simulated case of extravasation from the penile urethra. The skin has been removed from the penis and scrotum. The subcutaneous tissue has been incised in the midline and reflected laterally over the lower abdominal wall and along the dorsal shaft of the penis. The aponeurosis of the external oblique muscle is opened over the rectus abdominis muscle and down through the right inguinal canal (left side of illustration). The other layers of the rectus sheath have also been opened transversely and longitudinally near the umbilicus. Note that the yellow latex used to simulate the extravasate infiltrated the spermatic cord from the lower part of the scrotum and ascended retrogradely through the inguinal canal. Also note the simulated extravasate in the subcutaneous tissue along the shaft of the penis. Some of the latex also followed along the areolar tissue surrounding the superficial vessels and nerves and entered into the layers of abdominal muscles at the points where the vessels and nerves made their exits, as shown by the latex over the rectus sheath and on the aponeurosis of the external oblique muscle. The sac of an indirect inguinal hernia is shown at the left subcutaneous inguinal ring (right side of figure). ×0.2.

The importance of the connections of the membranous layer to the deep fascia and periosteum of the body wall, and its continuity from the perineum around the scrotum and penis and over the ventral abdominal wall, has been stressed in both anatomic and clinical texts because of its role: (1) in limiting and directing the course of extravasated urine, blood, pus or gases; (2) as a surgical guide in making drainage incisions; and (3) in the approximation of the edges of the membranous layer in surgical closure of incisions in the ventral abdominal wall and in the perineum. The presence of an outer, subcutaneous fatty layer in many parts of the body is accepted, but there is some doubt as to the presence and the continuity of a definite, deeper membranous layer (Scarpa's and Colles' fasciae), which is distinct from the outer fatty layer in the regions under consideration.

In the first place, there appears to be no established definition of, or priority for, the terms Camper's and Scarpa's fasciae. The original papers by Camper (1768 and 1801)[1] and Scarpa (1809),[2] which might possibly have been the source of these terms, dealt primarily with the anatomy and surgery of hernia and did not describe these layers as they are currently defined in the anatomic and clinical texts; but through some sources as yet unidentified and by repetition of erroneous text descriptions and illustrations their names have become associated with these layers.

In 1811, Colles described a dense fascial layer in the perineum but he did not state whether it was fatty or membranous in character nor whether it was attached to the posterior border of the urogenital diaphragm, as shown in the following quotation from his work:

"Now proceed to dissect the perineum. Raise the skin of the perineum, extending the dissection beyond the tubera ischii to the thighs. This exposes to view a strong fascia, which, on dissection, will be found to cover the entire of the perineum, and to blend itself with the structure of the scrotum. This fascia, although on a superficial view it appears continuous with the fascia of the muscles of the thigh, will yet be found, on closer examination, to attach itself very firmly to the rami of the ischium and pubis. The texture and connexions of this fascia will serve to explain many of those phenomena attendant on the effusion of urine into the perineum, by rupture or ulceration of the posterior part of the canal of the urethra. . . . First, then, you will find that this fluid, when so effused, although it forms a tumour in perineo, rarely terminates by suppuration and ulceration in this spot; being here resisted by the dense and unyielding texture of the fascia, diffusion laterally towards the thighs is prevented by the close attachment of this fascia to the rami of the pubis and ischium; while its progress forwards is favoured by a quantity of cellular substance, interposed between the surface of the perineal muscles and this fascia. . . . Before you raise this superficial fascia of the perineum, it may be of use to make a transverse incision through it, midway between the tuberosities of the ischia and the arch of the pubis. This incision will enable you to see the muscles of the perineum, lying in their natural situations. . . . Proceeding in the dissection, now remove the superficial fascia of the perineum, and then clear away some cellular substance covering the muscles, so as to give a distinct view of them. Next remove these muscles, taking care not to cut another fascia which lies under them, and which we shall presently examine, under the name of the anterior layer of the triangular ligament of the urethra, or membranous septum of the perineum."

Therefore, except in the case of Colles' fascia, original definitions are not given for the extent and connections of these fascial layers, and the fatty or membranous nature of all three fasciae is not established. It is interesting to note that Scarpa's and Colles' fasciae are not mentioned in Gray's *Anatomy* until 1897, while Camper's fascia was not defined until the 1901 edition.

Secondly, the indefiniteness as to the existence of the inner membranous layer is further evident from the accounts and illustrations given in the current texts and journals regarding the structure, position, and connections of this layer from an anatomic and clinical point of view.

Histologically the subcutaneous tissue, except in certain limited regions of the body devoid of fat like the eyelids or external ear, consists of a network of collagenous and elastic fibers, surrounding lobules of fat, which extend from the periosteum or deep fascia to

[1] A study of the available works by Petrus Camper on many phases of his medical research, as well as the *Oeuvres* de Pierre Camper (Paris, H. J. Jensen, 1803, 1: 17–103) and the commemorative papers on the 150th anniversary of his death (*Nederland. Tijds. Geneesk*, 1939, 33: 2085–2147) contain no mention of his name associated with a superficial fatty layer of subcutaneous tissue. The Dutch eulogists most likely would have given him credit for naming this fascia if he had done so.

[2] Sir John Struthers (1854) was the earliest source found to use the term Scarpa's fascia and then only for brevity of description rather than from authoritative definition (See: Tobin and Benjamin, 1944, for quotations from this paper).

the dermis. It is also now generally agreed that fat is formed within the fibroblast-like cells (Clark and Clark, 1940) of the collagenous-elastic network in the subcutaneous tissue and that the proportion of fat to fibers varies with the obesity of the individual and with the area of the body under consideration. Therefore, except in certain regions where the subcutaneous tissue is devoid of fat, a definite membranous layer is not found. Besides, fat cells do not exist individually or in groups without an embedding connective tissue framework (Bremer and Weatherford, 1944).

The current descriptions of these fascial layers found in the texts also do not agree with specific studies of the subcutaneous tissue. For example, Blechschmidt (1930) in making a study of the subcutaneous tissue from many regions of the body in infants and adults, based upon frozen sections stained with methyl blue or van Gieson stains and photographed by transillumination or reconstructed as waxed models, observed no subdivision of the subcutaneous tissue into an outer fatty and an inner membranous layer. Only one layer, consisting of fat embedded between strands of connective tissue, was found between the body wall and the dermis in many areas of the body. Congdon and associates (1946) employing relatively thin sections of the ventral abdominal wall, several inches in length, cut with a razor blade and stained with a fat stain (Sudan IV), also demonstrated only one layer composed of sheets of connective tissue around deposits of fat in the subcutaneous layer. Any one of the collagenous-elastic sheets within the fat could be called the membranous layer. A similar finding was reported by Forster (1937) studying the cut edges of incisions made into or through the subcutaneous layer of the ventral abdominal wall of cadavers. The fibrous strands or sheets which might be called the membranous layers were embedded within the subcutaneous fatty panniculus, with fat on both sides of the membranous sheets or strands. Other specific works dealing with the fascia of the human body, such as the one by Singer (1935), also show that membranous layers are embedded within the fat. Some texts even consider the inner membranous layer as the under or deepest surface of the tela subcutanea, as opposed to the preceding view (Davis, 1918; McGregor, 1946). Further confusion is added since it is also stated that the superficial lymph nodes or vessels, vascular channels, and cutaneous nerves run between Camper's and Scarpa's fascia, whereas other texts indicate that they course within Camper's layer. In the perineum, the posterior scrotal nerves and vessels are described as running deep to, superficial to, or within Colles' layer.

Although it is generally stated that extravasates in the perineum do not diffuse further dorsally than the limits of the superficial transverse perineal muscles (posterior part of the urogenital triangle), cases are recorded where extravasates were found along the sides of the anus in the ischiorectal fossa (Wolfer, 1918). The membranous layer is described as fused with the inguinal ligament or, immediately distal to this ligament, with the fascia lata of the thigh. However, extravasates (particularly blood) have been known to pass down on the front of the thigh distal to these connections. The presence of one subcutaneous layer—fatty and membranous components intermingled and not two separate and distinct layers—would help to account for these anatomical and clinical differences.

Except for the microscopic studies mentioned above, methods of definitely establishing the presence of these layers have been questionable. Grossly it is not possible in the majority of instances to demonstrate a definite inner membranous layer over the ventral abdominal wall or in the perineum of the normal cadaver or patient without creating such a layer artificially by dexterous use of the scalpel, as many anatomists and clinicians are well aware. The collagenous-elastic components of the subcutaneous tissue may be thickened, due to the resultant inflammatory process, when large amounts of extravasated urine, blood, pus or gases are present in this tissue, or these components may also be thickened by stress due to subjacent herniation or abnormal growths. In such instances a definite inflammatory or tensile membranous layer will easily be found—but usually with fat on each side of the layer.

A restudy of the subcutaneous tissue over the ventral abdominal wall, penis, scrotum,

and perineum was made in order to re-evaluate the anatomy and the clinical importance of Camper's, Scarpa's and Colles' fasciae, in view of: (1) the indefiniteness of the structure of the subcutaneous tissue; (2) the presence of a membranous layer (or layers) within or at the deep surface of this tissue; and (3) the connections of the membranous layer to the deep fascia or periosteum which would limit and direct the spread of extravasates and infections.

MATERIAL AND METHODS

The following methods of approach were utilized to study the subcutaneous tissue of these regions.

1. Microscopic sections were made after fixation in 10 per cent formalin or Zenker acetic solution: (a) from blocks of the hypogastric region of the ventral abdominal wall, including all the tissue from the peritoneum through the epidermis from 6 adult male cadavers of varying obesity, for the normal adult structure of the subcutaneous tissue; (b) from samples of the tissue, called "Scarpa's fascia," obtained from the abdominal wall of 5 patients at operation, for the surgical concept of this layer; and (c) from segments of the abdominal wall taken from 3 cases of urinary extravasation (from the penile urethra) to determine the tissue involved in this type of extravasation[3]. In most instances, these tissues were cut at 10 micra in paraffin and were stained with Heidenhain's modification of the Mallory connective tissue stain. Other samples of the same tissues were also cut in celloidin or with the freezing microtome and stained with hematoxylin and eosin, orcein (for elastic fibers), or the Heidenhain's connective tissue stain; in addition, Sudan IV or Sudan black B were used as fat stains on selected sections.

2. Macroscopic sections were also made since the amount of tissue which can be cut in paraffin and celloidin or with the freezing microtome is so limited and hence the continuity and attachments of the connective tissue layers is interrupted by these methods. These macroscopic sections were made by embalming the larger specimens with, or immersing the smaller ones into 10 per cent formalin and subsequently freezing them with dry ice (solid CO_2). Such specimens consisted of: (a) a portion of the body including the lower abdomen, pelvis and thigh from 5 full term, male fetuses which were cut on a Vaughan high speed band saw (3 cut in the sagittal and 2 in the coronal plane) as ¼ inch thick sections; (b) comparable regions from 5 adult males similarly prepared and cut (3 in the sagittal and 2 in the coronal plane) as ½ inch thick sections; (c) segments of the ventral abdominal wall, approximately 3 to 5 inches long and 1 to 2 inches wide, taken parallel to the mid-rectus incision distal to the umbilicus from 20 bodies at autopsy, including all the tissue from the peritoneum through the skin, were cut longitudinally as ⅛ inch thick sections; (d) similar blocks were taken at a right angle to the mid-rectus incision both above and below the umbilicus

[3] Brief histories of these 3 cases are:

CASE 1. G. K., S. M. H., No. 199108, a 60 year old, white male was admitted November 30, 1942 with symptoms of urinary extravasation and respiratory infection with cough of 8 days' duration prior to admission. His abdomen was reddened with subjacent swelling from the symphysis to the umbilicus, more marked on the right than the left side. His penis was gangrenous and tender; the scrotum was swollen, red, and tender. The patient was sent to the operating room immediately. The gangrenous areas of the penis were removed. Multiple incisions were made into the perineum, penis, scrotum, and abdomen, and drained. He died 7 days after admission with a clinical diagnosis of lobar pneumonia as the cause of death. The unclaimed body was subsequently sent to the Department of Anatomy for embalming and dissections. Samples of tissue were taken from the various areas involved in the extravasation for microscopic study. This case was also used in a previous study (Tobin and Benjamin, 1944).

CASE 2. E. G., R. M. H., No. 239224, a 45 year old, white male was admitted April 20, 1947 with chills and fever, pain in the groin, scrotum, and perineum which had been swollen and tender 4 days prior to admission. The next day his perineum, scrotum, penis, and lower abdominal wall were red and swollen with a typical outline of extravasation from the penile urethra, but he had no difficulty voiding. Multiple incisions were made into the subcutaneous tissue of the involved areas and drained. A sample of skin and subcutaneous tissue was taken an inch above the mid-point of the left inguinal ligament for microscopic study. A diagnosis of urinary extravasation with bacterial infection of these areas was made. There was sloughing of the tissues particularly over the scrotum which required secondary closure and prolonged his hospitalization. He was discharged as cured on June 1, 1947.

CASE 3. W. A., R. G. H., No. A 14333 R, a 74 year old, white male was admitted December 15, 1947 with pain and soreness in lower abdomen and incontinence for previous 24 hours. His penis was markedly swollen and gangrenous and leaking urine from a fistula on the ventral surface. His scrotum and perineum were red and distended, with involvement of the abdominal wall from the pubis to the umbilicus and laterally to the iliac crests and for an inch below the inguinal ligaments, as well as in the anterior part of the ischiorectal fossa, along the sides of the anus. A suprapubic cystotomy was done. Multiple drainage incisions were made into the subcutaneous tissue of the involved areas and drains were placed therein. A sample of the skin and subcutaneous tissue was taken, 1 inch distal and lateral to the umbilicus from the right side for microscopic study. There was considerable sloughing of skin and subcutaneous tissue from the involved areas, which required prolonged hospitalization. He had a similar extravasation about 12 years previously due to an injury of his perineum. He was discharged February 22, 1948, after unilateral orchiectomy and plastic repair of the penis.

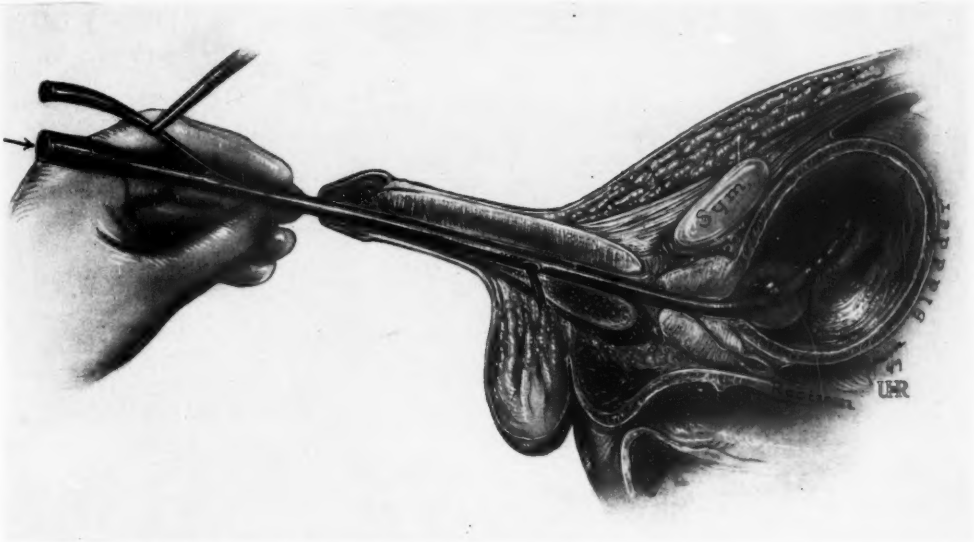

Fig. 1. Drawing to illustrate the method for producing simulated extravasation from the penile urethra. A sagittal section of the pelvis and perineum is shown for clarity. The penile urethra was visualized by means of a McCarthy panendoscope through which a fulgurating electrode was inserted to perforate the ventral wall of the urethra (at approximately 1 inch distal to the external sphincter), the corpus spongiosum urethrae, bulbocavernosus muscle and its covering of Buck's fascia. The panendoscope and electrode were withdrawn and a modified Foley catheter, with the "bladder openings" ligated and its lumen perforated to correspond to the level of fulguration, was then inserted through the penile urethra and into the bladder. The balloon was inflated and retracted against the bladder neck. The air passage of the catheter was compressed with a hemostat to keep the balloon inflated. Compressed air followed by a diluted rubber solution (red or yellow liquid latex) was then injected via the catheter into the tissues of perineum to simulate course of extravasation. Arrows indicate pathway of injection mass. 0.75 normal size.

from 2 adult male bodies and were prepared in the same manner as the sections from (c); and (e) 4 samples (1 to 2 inches wide and 3 inches long) of the skin, subcutaneous tissue and underlying muscle or fascia from the lateral surface of the thigh, arm, and back were similarly prepared to compare the structure of the subcutaneous tissue in these regions with that of the abdominal wall from the bodies used in (d). These macroscopic sections were stained with aniline blue to demonstrate the form, relations, and connections of the collagenous strands or bands, which make up most of the membrane-like layer, over relatively large areas of the subcutaneous tissue.[4]

3. Gross dissections of the regions under consideration were also studied in 30 adult male cadavers in the dissecting rooms, and photographs taken of the dissected subcutaneous tissue in some specimens to verify the conclusions drawn from the stained microscopic or macroscopic sections.

4. Cases of urinary extravasation were also utilized. In addition to the microscopic sections from the 3 real cases of extravasation mentioned before this type of extravasation was simulated in 10 cadavers by perforating the ventral wall of the penile urethra (approximately 1 inch distal to the external sphincter), the corpus spongiosum and its tunica albuginea, the bulbocavernosus muscle and its covering of Buck's fascia, by means of a fulgurating electrode introduced through the urethra via a McCarthy panendoscope to visualize and direct the depth of perforation. Following this simulated perforation, a Foley (24 or 26 French) catheter was inserted into the penile, membranous, and prostatic urethra and for a short distance into the bladder. This catheter had been modified before insertion so that the

[4]See: Tobin and Rumery (1948) for a more detailed account of the preparation and staining of such macroscopic sections.

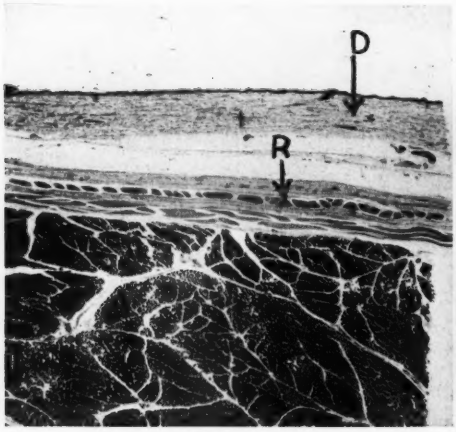

Fig. 2.

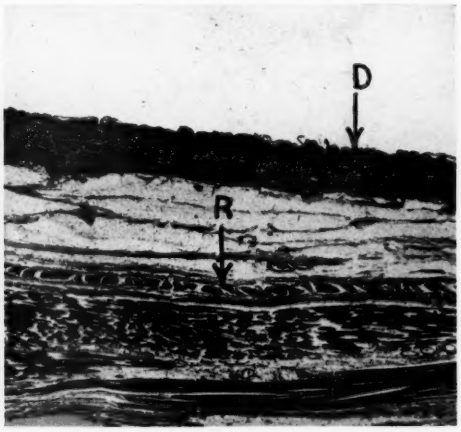

Fig. 3.

Fig. 4.

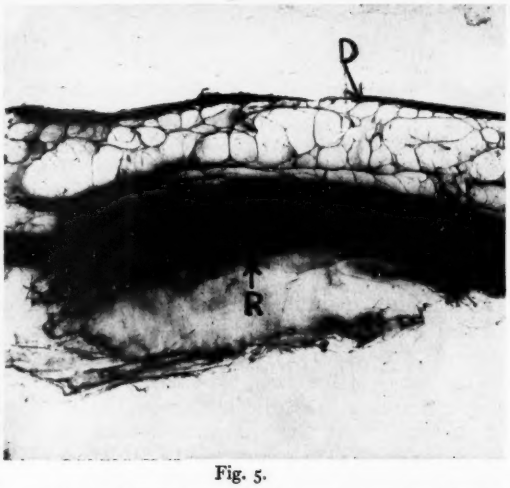

Fig. 5.

Fig. 2. Photomicrograph of a paraffin section through the abdominal wall, taken 1 inch lateral and distal to the umbilicus, from an emaciated adult male. The subcutaneous tissue consists of strands of collagenous and elastic fibers around deposits of fat (clear areas) between the dermis, *D*, and the sheath of the rectus abdominis muscle, *R*. The collagenous fibers in the inner part of the subcutaneous tissue immediately above the rectus sheath might possibly be interpreted as a membranous layer in this section. Compare with Figures 3 and 4. Heidenhain's connective tissue stain. ×9.

Fig. 3. Photomicrograph of a paraffin section, through the abdominal wall from an adult male of average obesity. Note the increased amount of fat (clear areas) between collagenous and elastic fibers and no suggestion of a definite inner membranous layer in the subcutaneous tissue. Site from which section was taken, the stain used, and the labels are the same as for Figure 2. ×9.

Fig. 4. Photomicrograph of a paraffin section, through the abdominal wall from an obese adult male. The laminated structure of the fat surrounded by collagenous and elastic fibers in the subcutaneous tissue does not suggest any division of this tissue into an outer fatty and an inner membranous layer. Site from which this section was taken, the stain used, and the labels are the same as for Figure 2. ×9.

Fig. 5. Photograph of a macroscopic, frozen section cut in the longitudinal plane through the pararectus region of the abdominal wall from an obese, adult male. Collagenous tissue was stained with aniline blue. Note that the subcutaneous tissue between the dermis, *D*, and the rectus abdominis muscle and sheath, *R*, consists of collagenous fibers around the alveoli of fat. As demonstrated in Figures 2 to 4, there is no subdivision of the subcutaneous tissue into an outer fatty layer and an inner membranous layer. ×0.6.

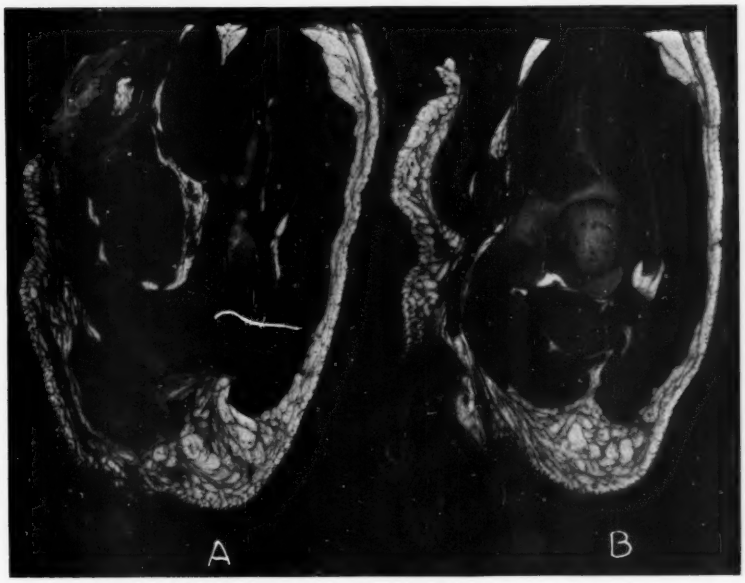

Fig. 6. Photograph of macroscopic, frozen sections cut slightly obliquely in the sagittal plane through the pelvic area of a full term male infant. Collagenous tissue in these sections was stained with aniline blue. Note the strands of collagenous fibers around the fat deposits in the subcutaneous tissue in both sections A and B, with no separation of this tissue into an outer fatty and an inner membranous layer over the abdominal wall or over the perineum. ×0.8.

bladder openings were ligated and other apertures made into the lumen of the catheter to correspond to the fulgurated perforation. The balloon was then inflated and retracted against the bladder neck with considerable force to prevent the injection mass from entering the bladder. Compressed air followed by an injection mass, red or yellow liquid latex, was then injected into the catheter so this mass could simulate the course of extravasation (Fig. 1). These cadavers were afterwards embalmed via the femoral artery with 10 per cent formalin. Subsequently 3 of these cadavers were studied by progressive stages of dissection to determine the layers involved in this simulated extravasation; whereas the lower abdomen, pelvis, and thigh of the other 7 were frozen in dry ice and cut into sections (1 to 1½ inches thick) on a band saw, either in the sagittal or coronal plane, to avoid interrupting the layers which result when dissection is used as a means of study. These sections were used to observe the course of the simulated extravasation.

OBSERVATIONS

Anatomical. There was no evidence of a subdivision of the subcutaneous tissue over the ventral abdominal wall and the perineum into an outer fatty (Camper's) and an inner membranous (Scarpa's-Colles') layer, for this tissue in both microscopic and macroscopic sections of newborn and adult male specimens consisted of a single layer composed of collagenous and elastic fibers[5] in the form of sheets or bands enclosing deposits of fat (Figs. 2 through 11). The subcutaneous tissue over the penis and scrotum consisted of a loose network of collagenous and elastic fibers in which isolated bundles of smooth muscle (dartos) fibers were embedded. The cutaneous nerves, superficial vessels, lymph nodes (Figs. 7 and 9) and vessels were embedded in this one common, continuous layer and not embed-

[5]Collagenous fibers made up the greater part of the sheets or bands enclosing the deposits of fat, although elastic and reticular fibers were also present in much smaller amounts. Therefore in the subsequent discussion, the term "fibers" will be used in reference to the collagenous fibers.

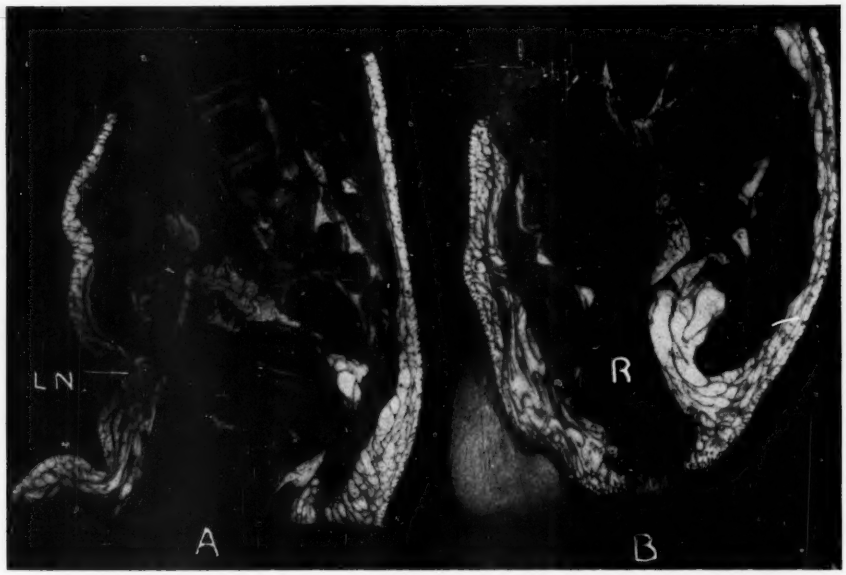

Fig. 7. Photograph of macroscopic sections from the same specimen shown in Figure 6 and cut and stained by the same methods. In A note the continuity of the superficial collagenous fibers in the subcutaneous tissue over the abdomen onto the thigh, whereas the deeper fibers are attached to the fascia lata; and in B the direction of the collagenous fibers up into the ischiorectal fossa around the rectum, *R*. Compare the direction of the latter fibers with those over the perineum shown in Figures 6 A and B. The location of the inguinal lymph nodes, *LN*, within the subcutaneous layer is shown. ×0.8.

ded within a fatty layer, between a fatty and membranous layer, or deep to a definite membranous layer in the lower abdominal, penile, scrotal, and perineal regions. In dissections of these regions, a cleavage plane could be made along any one of the several sheets or bands of fibers around the fatty alveoli in addition to the artificially created "membrane-like" layer in the inner part of the subcutaneous tissue (Figs. 12 and 13).

The subcutaneous tissue in these regions was continuous with that in adjacent areas of the body and had the same structure. However, the direction, the extent, and the attachment of the collagenous fibers to the deep fascia and periosteum of the body wall were related to the degree of movement possible in the overlying skin. Where considerable movement was possible, the course of the fibers was mainly parallel to the skin and in the form of laminae made up of strands or bands several inches in length (Figs. 5, 6 and 9). In such areas, only areola-like fibers attached the subcutaneous tissue to the subjacent body wall. In the midline distal to the umbilicus the deeper fibers were thickened to form the suspensory ligament of the penis. Cephalad to this ligament there was no evidence of a definite partition formed in the midline by a continuous attachment of fibers to the linea alba which would create two lateral compartments over the ventral abdominal wall to limit the spread of extravasates to either side exclusively. However, at the junction of the inferior extremity with the pelvis, the fibers were very firmly fused to the deep fascia or periosteum of the body wall. Many of these fibers were attached to the inguinal ligament or to the fascia lata of the thigh. A smaller superficial (subdermal) group of fibers surrounding deposits of fat continued in their course down the thigh (Figs. 7, 9, and 22). In the perineum the fibers were connected laterally to the periosteum of the inferior rami of the pubis and ischium (Figs. 10 and 22). There was a weak attachment of these fibers to the midline over

Fig. 8. Photograph of a macroscopic, frozen section cut in the coronal plane through the perineum of a newborn male infant. Note the arrangement of the collagenous strands stained with aniline blue between the components of the penis, *P*, and the skin of the perineum. There is no definite layer which could be designated as a membranous (Colles') layer. ×0.9.

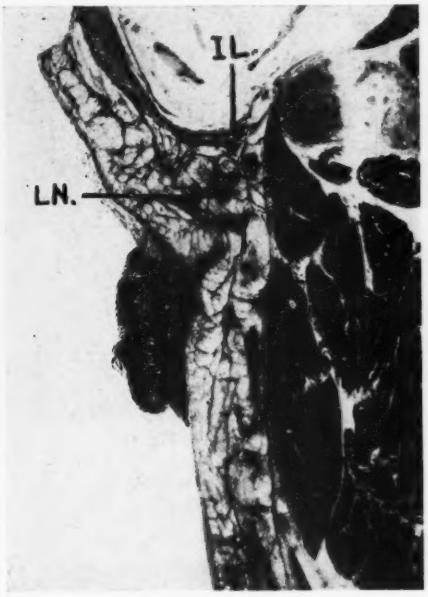

Fig. 9. Photograph of a macroscopic, frozen, parasagittal section, through the pelvis and thigh of an adult male. Collagenous tissue stained with aniline blue. Note the structure of the collagenous fibers around the deposits of fat in the lower abdominal region and the continuity of the superficial fibers on the thigh; whereas many of the deeper fibers are connected to the fascia lata near the inguinal ligament or directly to this ligament, *IL*. Superficial nerves, blood vessels, lymph nodes, *LN*, and vessels are embedded within this single layer of fat and collagenous fibers and not between an outer fatty and an inner membranous layer. ×0.18.

Buck's fascia covering the bulbocavernosus muscle but this attachment was not as firm as that to the rami of the pubis and ischium. At the posterior border of the urogenital triangle the majority of these fibers coursed cephalad into the ischiorectal fossa and were at approximately a right angle to those superficial to the Buck's fascia over the 3 components of the penis in the perineum (Figs. 6, 7, 8, 10, 11, and 22). There was no evidence that these fibers attached to the posterior border of the urogenital diaphragm.

Clinical. The finding of one and not two separate (outer fatty and inner membranous) layers of subcutaneous tissue in the anatomic observations was verified from sections of the tissue designated as the membranous (Scarpa's) layer removed from the abdominal wall at operation. Such tissue consisted of strands or sheets of collagenous fibers embedded in or surrounding fat (Fig. 14) and not a distinct membranous layer. Sections of the skin and subcutaneous tissue from the 3 cases of urinary extravasation also showed that the extravasate was within the subcutaneous laminae, formed by the sheets or strands of fibers around the lobules or alveoli of fat (Figs. 15, 16, and 17), and not limited by an inner membranous layer nor deep to the whole subcutaneous layer—between it and the intrinsic fasciae covering of the muscles of the body wall. In some instances extravasates may probably be found in such a location, but from these 3 real cases and the 10 simulated cases of extravasation it seems unlikely that this is the usual pathway; for in all the simulated cases, the injection mass was within and not deep to the subcutaneous layer (Figs. 18, 19, 20, and 21). The extravasate followed the course of the fibrous sheets or bands, around the fat deposits, and infiltrated the subcutaneous layer from the body wall to the dermis (Figs. 15 through 21).

The loose "areola-like" layer of collagenous and elastic tissue in the scrotum and penis was infiltrated readily with the injection mass (Figs. 18, 19, 20, and 21). This mass was also found in the subcutaneous tissue between the

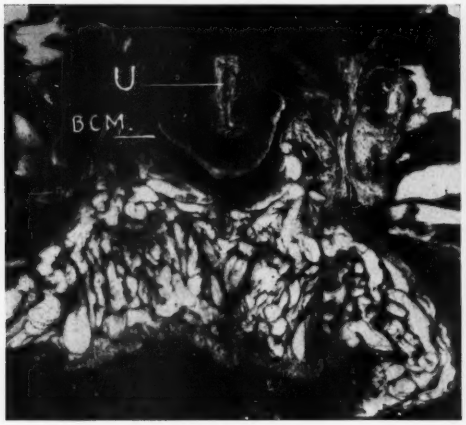

Fig. 10.

Fig. 11.

Fig. 12.

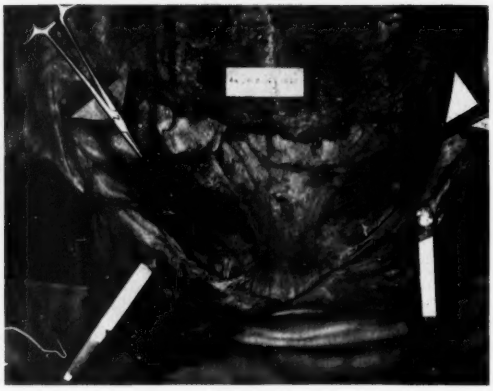

Fig. 13.

Fig. 10. Photograph of a macroscopic, frozen section, cut in the coronal plane through the perineum of an adult male. Collagenous fibers are stained with aniline blue. Note the absence of a definite membranous layer between the bulbocavernosus muscle, *BCM*, around the corpus spongiosum urethrae containing the urethra, *U*, and the skin of the perineum. Many of these collagenous strands connect to the periosteum of the inferior rami of the pubis and ischium. There is a loose attachment of the collagenous fibers to Buck's fascia covering the bulbocavernosus muscle. ×0.88.

Fig. 11. Photograph of a macroscopic, frozen section cut slightly obliquely in the coronal plane through the ischiorectal fossa of the same specimen shown in Figure 10. Note the cephalic direction of the collagenous strands stained with aniline blue around the deposits of fat in the ischiorectal fossa on each side of sphincter ani externus muscle, *S*. These strands are approximately at a right angle to those collagenous strands which lie over the components of the penis in the urogenital triangle. Compare with Figures 8 and 10. ×0.44.

Fig. 12. Photograph of a dissection of the subcutaneous tissue over the ventral abdominal wall showing the inner membranous layer (Scarpa's fascia) dissected by the students in gross anatomy. The skin has been reflected and the usual concept of the superficial fatty layer (Camper's fascia) dissected from the inner membranous layer and reflected laterally. Hemostats are elevating the "membranous layer" from the deep fascia over the aponeurosis of the external oblique muscles. The suspensory ligament of the penis is shown in the midline between the two reflected flaps of the "fatty layer." ×0.25.

Fig. 13. Photograph of the same specimen shown in Figure 12. Hemostats are elevating the dissected "inner membranous" layer but an additional membranous layer, superficial to that dissected by the students, is shown over the handles of the forceps in the lower part. Three such membranous layers were found in this specimen of average obesity, indicating that dexterity with scalpel can create membranous layers in different depths of subcutaneous tissue. Compare with Figures 5 and 9 for collagenous strands which may be utilized as cleavage lines in dissection. ×0.25.

Fig. 14. Photomicrograph of the tissue designated as Scarpa's fascia, removed from the subcutaneous tissue approximately an inch cephalad to the midpoint of the inguinal ligament, during repair for inguinal hernia in an adult male. This sample of Scarpa's fascia consists of layers of collagenous fibers surrounding fat and blood vessels and is not a distinct membranous layer. Heidenhain's connective tissue stain. ×9.

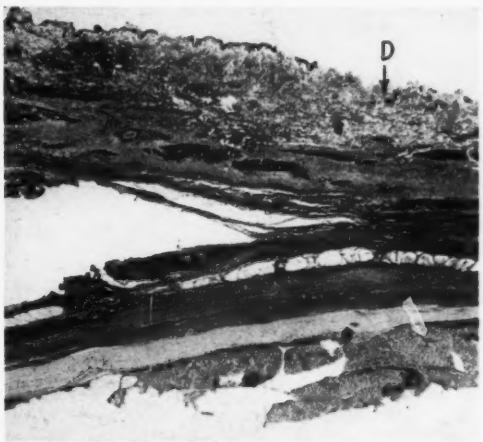

Fig. 15. Photomicrograph of a section through the ventral abdominal wall approximately an inch cephalad to the midpoint of the inguinal ligament from the embalmed cadaver of an adult male who had urinary extravasation from the penile urethra (See Case 1 in footnote). This section was cut on the freezing microtome and stained with Sudan black B to show the deposits of fat in the necrotic tissue on each side of the path of extravasation. The extravasate was not deep to a membranous (Scarpa's) layer but within the one common layer of subcutaneous tissue. The gap in the left side of this section is part of the original drainage incision in the subcutaneous tissue. Part of the epidermis was sloughed off. Note its absence over the dermis, *D*, in certain areas. The aponeurosis of the external oblique muscle is labelled, *E*. ×6.5.

dermis and Buck's fascia along the shaft of the penis, and in the subcutaneous layer over the ventral abdominal wall. Except in 1 instance the simulated extravasate did not extend further distally than 1 inch below the inguinal ligament. In several cadavers it did extend into the superficial part of the subcutaneous tissue around the anus, but not more than a half inch into the ischiorectal fossa (Fig. 18).

It is well known that extravasates through the penile urethra entering the vascular corpus spongiosum urethrae introduce urine, bacteria, and other matter into the vascular system. Subsequent infiltration of the subcutaneous tissue occurs after the extravasate has spread through the coverings of the corpus spongiosum urethrae and Buck's fascia. These studies with simulated extravasation, wherein the colored injection mass imitated the course of the urine, indicate that the extravasate may follow additional courses which could not readily be visualized in true cases of extravasation, except those of several days' duration in which local inflammation of the tissue would delineate the course. For example, as the simulated extravasate courses within the subcutaneous layer, it may infiltrate the loose areolar tissue around the walls of cutaneous vessels and nerves and follow retrogradely along their course to enter between the layers of the abdominal wall. The point of attachment of the testes to the lower part of the scrotal sac also consists of loose areolar tissue. The simulated extravasate, in 8 of the 10 experimental extravasations, was found to pass through this areolar tissue and to enter within the layers of the spermatic cord. Via this route, it coursed retrogradely upward within the spermatic cord (Fig. 20), through the inguinal canal in cases where inguinal hernia had distended the canal (Fig. 21), and infiltrated the retroperitoneal tissue of the abdomen.

In many clinical cases, following rupture of the mucous membrane in the penile urethra, the vascular, erectile tissue of the corpus spongiosum urethrae may be infiltrated with the extravasate before subsequent rupture of the tunica albuginea, bulbocavernosus muscle, and its covering of Buck's fascia result in infiltration of the subcutaneous tissue in this area. In other cases, the mucous membrane of the penile urethra may form a sinus tract through the erectile tissue and its coverings. The extravasate in such cases would involve the subcutaneous tissues only and by means

Fig. 16, left. Photomicrograph of a paraffin section of the skin and subcutaneous tissue removed from the area immediately above the left inguinal ligament from a patient with urinary extravasation from the penile urethra (See Case 2 in footnote) at the time of surgical drainage of the areas of extravasation. Note from the dermis, *D*, inward, the extravasated urine, bacteria, and blood cells followed the course of the collagenous fibers surrounding the alveoli of fat as shown by the dark stained material along the fibers. Heidenhain's connective tissue stain. ×8.

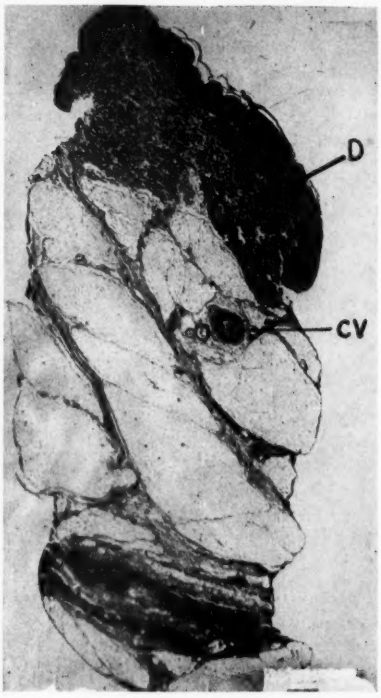

Fig. 17. Photomicrograph of a paraffin section of the skin and subcutaneous tissue removed from an area 1 inch lateral and distal to the umbilicus from the right side of a patient at the time of surgical drainage of the areas involved in urinary extravasation from the penile urethra (See Case 3 in footnote). Note as in Figure 16, the extravasate followed along the collagenous fibers surrounding the deposits of fat and extended to under surface of dermis, *D*. Also note position of cutaneous vessels, *CV*, within subcutaneous fat. Heidenhain's connective tissue stain. ×8.

of this tract by-pass the erectile tissue. To test whether the method used in the simulated extravasation studies would involve the same tissues as those in the clinical cases with sinus tract formation, liquid latex was injected directly into the subcutaneous tissue over the perineal components of the penis in 2 cadavers. This injection mass was found to follow the same pathways as those described above for the simulated extravasations from the perforated penile urethra. In other studies, the injection mass when injected into the subcutaneous tissue above the pubic symphysis likewise filled the same areas in 2 cadavers. Finestone (1941) has made similar studies using a radiopaque injection mass and found the tissue involved to be the same as we have described.

DISCUSSION

Although the subcutaneous tissue in most regions of the body is described as consisting of only one layer, that in the region of the low-

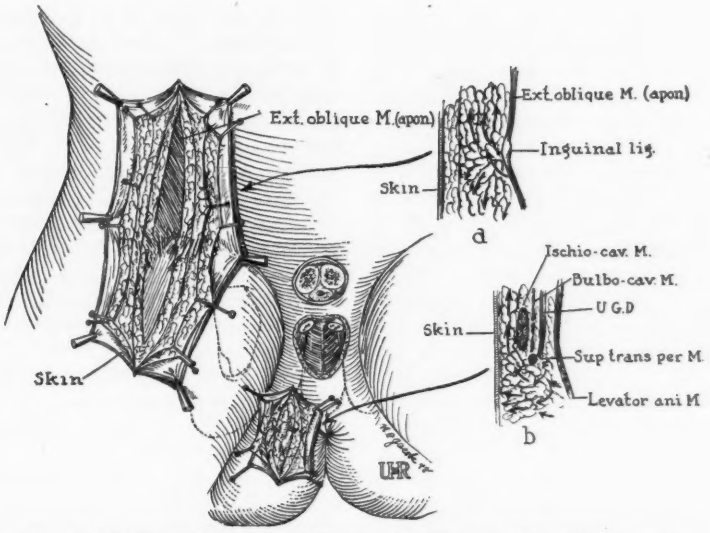

Fig. 22. Diagrammatic drawing to illustrate the structure of the subcutaneous tissue over the lower abdominal wall, proximal part of the thigh, and the perineum. For simplicity, the shaft of the penis and the scrotum are shown only as cross sections through their bases. The skin and the layers which can be dissected in the subcutaneous tissue proximal and distal to the inguinal ligament are shown in the left side of this diagram. A cross section from the same area is shown in insert a. Note, as shown by the arrows in the insert a, and the lines in the dissected area, that the majority of the fibers around the deposits of fat pass from one lamina (of fat and fibers) to the next subjacent layer. The fibers are attached to the inguinal ligament or to the fascia lata distal to this ligament. A smaller, finer group of fibers and fat, immediately beneath the skin continue directly from the lower abdominal area onto the thigh.

The composition of the subcutaneous tissue in the perineum is shown by a dissection and a cross section (insert b). In the dissection several layers could be created in the subcutaneous tissue and, as indicated by the arrows in the insert b, the majority of the fibers course parallel to the skin and the components of the penis in the urogenital triangle of the perineum. However, these fibers are continuous with similar fibers in the ischiorectal fossa of the anal triangle, but in the latter location they run at approximately a right angle to those over the urogenital triangle. These fibers did not have a definite attachment to the superficial transverse perineal muscles nor to the posterior border of the urogenital diaphragm, *U.G.D.* There was no evidence of a division of the subcutaneous tissue in the perineum, thigh, or lower abdomen into a definite outer fatty and inner membranous layers. Compare with Figures 9, 10, and 11 for the structure of the subcutaneous tissue in these areas as seen in stained sections. ×0.32.

er abdominal wall and in the perineum has been thought to be subdivided anatomically and clinically into an outer fatty and an inner membranous layer. It is interesting to note that approximately 90 years elapsed after Camper, Scarpa, and Colles published their respective works, from which the terms now used for the layers of the subcutaneous tissue in these regions might have been taken, before their names were introduced into the anatomic and clinical texts. Before that time, most of the texts stated that only one layer was present and only a few years before these proper names were introduced in the texts, were two layers assumed to be in the subcutaneous tissue. It is most likely if these layers were present, anatomists and clinicians at that time would have observed them. The evidence presented in this present work indicates that the older idea of one subcutaneous layer in the regions involved in extravasates from the penile urethra should be adopted again in keeping with the same structure of this tissue in other parts of the body.

The question may be raised, what governs and directs the classical spread of such extravasates if there is only one layer present in the perineum, scrotum, penis, and abdominal wall, which is continuous with the general subcutaneous layer in other parts of the body. The attachments of the collagenous fibers in the subcutaneous tissue to deeper structures at certain areas (such as the inguinal ligament, or adjacent fascia lata—Figs. 9 and 22—and the inferior rami of the pubis and ischium—Figs. 10 and 22—together with changes in direction of these fibers in certain areas, as in the ischiorectal fossa—Figs. 6, 7, 8, 10, 11, and 22) can account for the limitations. Differences in the structure of this tissue (areolar in the scrotum and around the penis, and fibrous strands surrounding fat in the form of loose laminae parallel to the surface of the skin over the perineum—Figs. 6, 8, and 10—and ventral abdominal wall—Figs. 2, 3, 4, 5, 6, 7, and 9) can account for its ease of spread in the typical directions. In some cases extravasated blood from injury in the perineum has been known to follow not only this typical course but also to extend into the subcutaneous tissue over the thigh and posteriorly along the sides of the rectum in the superficial part of the ischiorectal fossa.

Drainage incisions for extravasates are usually made to a depth which penetrates the whole of the subcutaneous layer. Yet it is known that in many instances the extravasate may be encountered in the superficial part of the subcutaneous tissue once the dermis is incised, and such material may be found within the entire thickness of the subcutaneous tissue; although in certain early cases the extravasate may be mainly in the deeper part of this tissue, before infiltration of the more superficial part has occurred. In the closure of incisions in these areas the entire depth of the subcutaneous tissue does not have to be entered before collagenous and elastic fibers are encountered, for they are found even in the superficial part of this so called fatty layer (Figs. 2, 3, 4, 5, 16, 17, and 22).

As pointed out above, unless the case of true extravasation is one of comparatively long standing wherein the inflammation within this tissue would outline the course of the extravasate, little can be known of its exact or probable extent. The information gained from these simulated extravasation studies suggests that the extravasate may involve other tissues than those usually described in real cases of extravasation. Extravasates may also course retrogradely within the areolar tissue along the sides of the superficial vessels and cutaneous nerves to enter between the layers of the abdominal wall, or within the layers of the spermatic cord; in addition to passing within the superficial part of the subcutaneous tissue down the front of the thigh or in the ischiorectal fossa.

The concept of one layer of collagenous and elastic fibers around deposits of fat and not two distinct layers (an outer fatty and an inner membranous) in the subcutaneous tissue has been recognized for many years. Langer (1861), Kocher (1907), and Cox (1941) suggested a similar structure for the subcutaneous tissue and showed that the cleavage lines of the skin are related to the direction of the fibers in this tissue as well as their connection to the deep fascia or periosteum in certain areas.

There appears to be no justification for the concept of an outer fatty (Camper's) and an inner membranous (Scarpa's-Colles') fascia in the subcutaneous tissue from historical priority of definition, anatomic structure, or the involvement of this tissue in real or simulated cases of urinary extravasation. This erroneous concept was introduced into the medical literature through sources as yet unidentified and has been perpetuated by tradition and inadequate means of study. The authors (Tobin and Benjamin, 1944 and 1946) also conformed to the usual concept of the form and attachments of these layers when dissection was used as a means of approaching such problems.

SUMMARY

The present concept of an outer fatty (Camper's) and an inner membranous (Scarpa's-Colles') fascia in the subcutaneous tissue over the ventral abdominal wall and in the perineum, and their role in directing and limiting the spread of extravasates are not justified for the following reasons:

1. There are many inconsistencies in the anatomic texts as to the structure and continu-

ity of these layers and their relation to the cutaneous nerves and vessels, which may be due to the fact that no priority of definition could be found for these fasciae since Camper, Scarpa, and Colles did not describe these layers as they are currently defined.

2. Studies from microscopic and macroscopic sections from these regions show that the subcutaneous tissue is made up of only one layer—strands or sheets of collagenous and elastic fibers surrounding deposits of fat which is similar to the structure of this tissue in other areas of the body.

3. Sections taken from, and dissections made of, real and simulated cases of extravasation from the penile urethra demonstrate that the extravasates are within the one common layer and not limited by a deep membranous (Scarpa's-Colles') layer. The attachment of the collagenous and elastic fibers to the deep fascia or periosteum of the body wall, as well as the change in direction of these fibers, and the compactness or looseness of the one subcutaneous layer account for the direction and limitation of the spread of extravasates.

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GASTRIC RESECTION FOR ESOPHAGITIS AND STRICTURE OF ACID-PEPTIC ORIGIN

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IN September 1939, a patient was operated upon by one of us, (O.H.W.), at the University of Minnesota Hospitals, for massive hemorrhage from a duodenal ulcer. The patient had an antecedent story of esophagitis, for which one of us (N.L.L.) had supervised frequent esophageal dilatations over a period of approximately 4 years. An unexpected windfall from this operation was that dilatation of the esophageal stricture became unnecessary following the procedure of gastric resection for the duodenal ulcer. In the course of the operative procedure, undertaken to control the gastric hemorrhage, simultaneous extensive gastric resection was performed to thwart further ulcer difficulty. Three years later, the same sequence of events was observed in another patient undergoing gastric resection for a gastrojejunal ulcer (following gastrojejunostomy performed for an obstructive duodenal ulcer); this patient also had esophagitis necessitating esophageal dilatation. On the basis of the cessation of necessity for continued esophageal dilatation in the first patient whose esophageal stricture had been dilated more than 100 times, one of us (O.H.W.) ventured to predict that successful relief of the ulcer diathesis in this instance too might make further esophageal dilatation unnecessary. Such proved to be the case.

When, therefore, a third patient presented herself with a picture of a congenitally short esophagus, an esophageal stricture, and gastric hemorrhage of unknown etiology, it was reasoned that the stricture in this instance too might be occasioned by the regurgitation of hydrochloric acid into the lower reaches of the esophagus. This latter patient was referred for resection of the esophageal stricture. She was in very poor physical condition, presenting all the clinical evidences of hypoproteinemia from starvation. The patient was easily persuaded, in the light of the success in the other 2 patients, to accept the suggestion that an extensive gastric resection be done with the thesis in mind of reducing the capacity of the residual gastric pouch to provide an active acid-peptic digestive juice, the regurgitation of which into the esophagus, in all likelihood, had been causing the patient's symptoms. A dramatic improvement followed the gastric resection and helped confirm in our minds the impression that esophagitis and esophageal stricture might well be a consequence of the acid-peptic digestive mixture in the same manner that duodenal ulcer is. Thereafter 2 additional patients with strictures fairly high in the esophagus were persuaded to accept the same operative procedure with hope of relief from esophageal obstruction. Operation in these latter patients has been done quite recently; yet the improvement has been so startling as to warrant, despite this somewhat meager experience, the placement of these case histories on record. In the main, they give fairly satisfactory proof that esophagitis is probably a manifestation of ulcer just as much as is an ulcer in the stomach or in the duodenum. Four of the patients listed herein probably had what is ordinarily called esophagitis. Three of the 4 had duodenal ulcer (Cases 1, 2, and 6) and rather severe concomitant complications from the duodenal ulcer; the fourth patient had esophagitis only. One of the remaining 2 patients had a congenitally short esophagus with a stricture. She too, however, had gastric hemorrhage. In this latter instance, prior to operation, the interpretation put upon the sequence of events was that she "probably had portal hypertension, a

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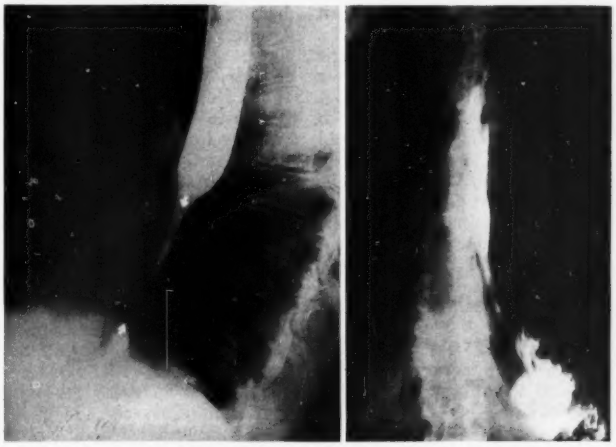


Fig. 1, Case 1. a, left, Esophagram showing extent of esophageal stricture, April 1, 1936; there is considerable dilatation of the esophagus above the stricture. b, Normal esophagram, April 3, 1948, almost 9 years after gastric resection, September 2, 1939, for bleeding duodenal ulcer. The patient is symptomatically well and has no demonstrable esophageal narrowing; moreover, there is no dilatation of the esophagus above the site of the former stricture.

ruptured esophageal varix which healed with stricture formation"—all of which suggested to the medical service that local excision of the esophageal stricture was perhaps the best therapy. The remaining patient in the group was a young boy who, since early years, had had trouble in swallowing; the difficulty in swallowing became progressive, eventually necessitating frequent dilatation. The case histories, of these patients, follow.

CASE HISTORIES

CASE 1. Mr. L. W. was admitted to the University Hospital on September 3, 1939 and dismissed on September 22, 1939. Patient was 53 years of age at the time of that admission (U. H. No. 643715).

Twenty years ago the patient had had a diagnosis of duodenal ulcer; however it did not give him much trouble until 1935, since which time he has had considerable trouble with periodic obstruction as well as bleeding. The patient was first admitted to the University Hospital in November 1935, with a diagnosis of duodenal ulcer with obstruction; he was hospitalized by the medical service for approximately 1 month and was placed on a Sippy regimen. Following dismissal he began reporting to the out-patient clinic of the department of medicine in January, 1936; in February 1936, he complained of difficulty in swallowing, and when an esophagram was made a stricture was described in the lower third of the esophagus. On esophagoscopy it was found to be 38 centimeters from the incisor teeth. The stricture was rather long and smooth. In April, 1936, dilatations were begun, and once or twice each month the esophagus was dilated up until the time of surgery, which was done in September, 1939. In the interval between dilatations, the esophageal stricture would usually shrink down to a No. 37 French but could be dilated again without too much difficulty up to a No. 45 French. In February, 1936, the patient was hospitalized again for the treatment of his ulcer for a period of about a month. He was discharged with the diagnosis of obstructive duodenal ulcer and esophageal spasm and stricture (Fig. 1, a). In September, 1936, he was hospitalized for a period of 3 weeks under medical management with the same diagnosis. In March, 1938, he was hospitalized again for a period of 2 weeks.

In August, 1939, the patient was admitted to the medical service for treatment of a bleeding duodenal ulcer. While under medical management the bleeding continued, and on September 2, 1939, the hemorrhage was profuse. The hemoglobin, despite transfusions, had fallen from 100 per cent following transfusions down to a level of 20 per cent. Because of this, operation was undertaken as an emergency measure, a two-thirds gastric resection being done with open closure of the duodenum. Considerable blood was found in the intestine at the time of the operation. Prior to operation the patient had 38 degrees free hydrochloric acid in a fasting specimen, without histamine. Convalescence was satisfactory and patient was dismissed from the hospital 20 days after operation. Several transfusions were given just prior, during, and after operation.

In November, the esophagus was dilated and it was found that it had only contracted down to No. 41 French and dilatation with a No. 45 French bougie was easy. A similar dilatation was done in December and the figures were the same. In February, 1940 it was found, on examination, that a No. 43 French catheter passed easily. The esophagus was dilated up to a No. 45. This was repeated in April and again in June, and when the bougie was passed in August, 1940, it was found that a No. 45 went down readily. The patient came in again for examination in March, 1941, at which time a No. 45 French sound passed easily. In January, 1945, 4 years later, the patient came in for observation, at which time he said he had had no further difficulty in swallowing and was not in need of further esophageal dilatation. He was seen here recently (April 1948); there was no difficulty in swallowing; an esophagram showed a fairly normal esophagus and a No. 45 French bougie passed without difficulty (Fig. 1, b).

Summary. A patient of 53 had been hospitalized many times because of the complications of duodenal ulcer. Prior to September, 1939, when an emergency gastric resection was done for massive hemorrhage from a duodenal ulcer, the patient's esophagus had been dilated approximately 100 times because of esophagitis and stricture. Whereas it had been anticipated that the need for esophageal dilatation would continue, it was observed that further esophageal dilatations were unnecessary (Fig. 1, a and b).

CASE 2. Mr. C. V. D. was aged 85 at the time of admission to University Hospital (U. H. No. 620411).

This patient was first seen at the University Hospital for prostatic obstruction in 1933, at which time he was 75 years of age. In July, 1934, he returned because of a very large right anterior suprahepatic abscess; the patient at this time was very ill. Aspiration with a needle was carried out below the right costal margin, the abscess being large and anterior. Subsequently an empyema trochar was inserted and a catheter was threaded into the abscess cavity. The patient had a duodenal fistula which closed only slowly. When subsequently during his convalescence, barium was given, the barium found its way through the duodenal ulcer perforation into a huge subphrenic space on the right side.

In 1938 the patient returned because of a bleeding ulcer. He was given several x-ray examinations during 1935 and 1936, and a definite duodenal deformity was observed. In 1936 he was hospitalized by the medical service because of hemorrhage and pain. In 1938 he was hospitalized again because of hemorrhage and at the same time he was treated on the eye service for a conjunctivitis of the left eye. Because of repeated and recurrent attacks of pyloric obstruction, the patient was hospitalized in December, 1941, and a gastrojejunostomy, together with an enteroanastomosis was made. The patient was 83 years of age and it was believed that because of his age, gastrojejunostomy was an adequate operation. He improved considerably after the gastrojejunostomy, but in January, 1942, he complained of a good deal of trouble in swallowing. Because of his generally poor condition he was readmitted for dilatation of the esophagus. Already in December, 1941, at which time the patient was admitted for gastrojejunostomy, narrowing in the lower end of the esophagus was noted on x-ray examination though at this time, the patient did not complain of much difficulty in swallowing. The patient continued to come to the out-patient clinic for periodic dilatation of the esophageal stricture involving the distal 4 centimeters of the esophagus. The patient at this time was subjected to dilatation at 2 week intervals. In August, 1942, it was suggested by one of us (O.H.W.) that gastric resection might help the esophageal stricture on the basis that it had helped the esophageal stricture in the instance of Case 1, Mr. L. W., who had undergone gastric resection 3 years previously for massive hemorrhage from a bleeding duodenal ulcer. Because of roentgen-ray evidence suggesting the presence of a jejunal ulcer as well as continued pyloric obstruction, gastric resection was undertaken. Because of the difficulty of mobilizing the deformed duodenum, a Finsterer exclusion type of operation called the "IVA operation" in this clinic was done. The weight of the fragment of stomach removed was 385 grams; obviously there was a good deal of pyloric obstruction to produce a stomach of this size. By this time, too, the technique of gastric resection for ulcer had been well standardized in this clinic, and the conventional short loop posterior anastomosis with a 75 per cent resection was done. The enteroanastomosis was taken down and an end-to-end anastomosis in the jejunum was made there.

Following the gastric resection the patient continued to improve quite definitely. Occasional esophageal dilatation was carried out but it soon became apparent that further dilatation was probably not necessary. On each occasion when dilatation was carried out after June, 1943, a No. 45 dilator passed through easily. When the patient was last seen in this clinic in February, 1945, it was obvious that further esophageal dilatation was unnecessary. A recent letter (May, 1948) from the patient's physician, Dr. M. D. Mosby of Long Prairie, Minnesota, states that the patient had no further difficulty in swallowing after the gastric resection and that the patient died of senility and uremia on July 3, 1947, at 90 years of age.

Summary. A patient of 85 years, since the age of 75, had been incapacitated because of frequent recurrence of complications of a duodenal ulcer. Gastrojejunostomy carried out for an obstructive duodenal ulcer was followed by a gastrojejunal ulcer, accompanied by pro-

gressive symptoms of esophageal stricture. Complete relief of the ulcer diathesis followed gastric resection and esophageal dilatation became unnecessary. The patient died 5 years later of senility at the age of 90 years; he had experienced no difficulty in swallowing after gastric resection.

CASE 3. Mrs. E. K., 58 years of age at the time of admission to the University Hospital (U. H. No. 672670).

This patient has been coming to the University Hospital intermittently since October, 1938. She then gave a story of dysphagia of 3 months' duration. In January, 1936, the patient had a massive hematemesis followed by melena. An abdominal exploration was done elsewhere shortly after an episode of hematemesis, at which time a large mass was found in the upper abdomen, and a diagnosis of cystic disease of the liver and pancreas was established. Subsequent to the operation two subsequent episodes of hematemesis were experienced by the patient. Large abdominal masses were observed here on physical examination; moreover, the patient was found to be anemic, the hemoglobin being only 63 per cent. The white blood cell count was normal with a normal differential. Red blood cells were 2,890,000. Plasma proteins, blood cholesterol and nonprotein blood nitrogen values were normal when the patient was first seen here. Liver function tests were within normal limits.

X-ray examinations demonstrated a stricture in the lower 10 centimeters of the esophagus. The suggestion of a congenitally short esophagus was made. Esophagoscopy revealed a stricture 31 centimeters from the incisor teeth. In the x-ray film the stricture appeared to cover a distance of 3 to 4 centimeters. A pebble-like appearance caused by the inflammation of the dilated portion of the esophagus was reported.

Because of the stricture, dilatations were instituted beginning with a No. 31 French bougie; the first dilatations were carried up to a No. 39 French. The patient was dismissed from the medical service with a diagnosis of esophageal stricture, "probably on the basis of a ruptured esophageal varix with secondary scarification of the esophagus."

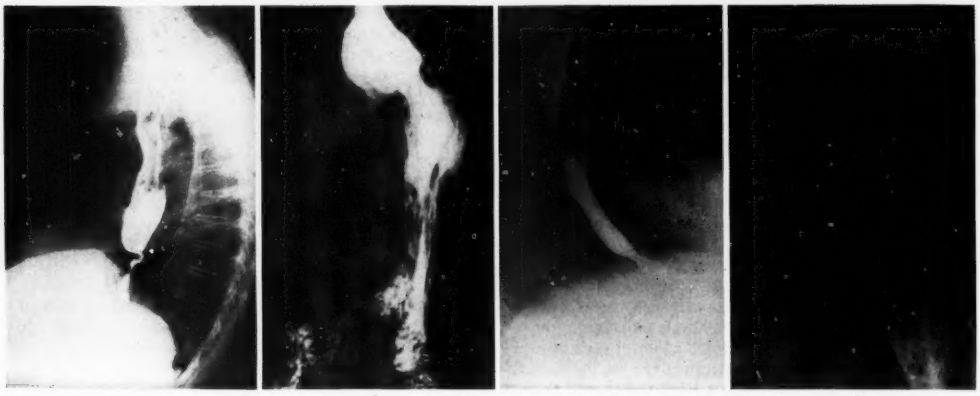
Over a period of the next few years the patient was admitted frequently to the medical service of the University Hospital here for dysphagia, hematemesis, or other circumstances requiring hospitalization. On one hospital admission a cystocele was repaired; the patient intermittently had swelling of the ankles; there was some hypertension with electrocardiographic evidence of myocardial damage, probably the result of an earlier myocardial infarction. The blood pressure varied between 150 and 185 systolic, with diastolic recordings in the area of 90 to 100. Passive congestion of the lungs and liver was diagnosed. The patient also had emphysema. The plasma proteins were quite consistently under 6 grams per cent, and on one occasion the plasma proteins were as low as 5 grams per cent.

Gastric analysis was made on several occasions, and free acid without histamine was 28 degrees with a total acid of 54 degrees; on another occasion with histamine the free acid was 34 degrees with a total of 50 degrees. However, the recovery in quantity of acid from the stomach was usually large on each occasion. The patient had occult blood in the stool quite consistently, and the hemoglobin varied between 7 and 11 grams.

Despite monthly dilatation the patient's nutrition seemed to worsen, and because of the difficulty in swallowing and the fairly persistent anemia, and repeated episodes of bleeding, the patient was referred for excision of the stricture in the esophagus in April, 1944 (Fig. 1, a). The patient lived in the northern section of the state, almost 400 miles from Minneapolis and had been contemplating moving her home to this area to lessen the difficulty of travel made necessary by the frequent dilatations. The stricture began apparently in the upper portion of the lower third of the esophagus, and because of the patient's poor condition, operation at that time was not recommended. However, the patient was advised that the patient in Case 1 reported here too had undergone approximately 100 esophageal dilatations, but that complete freedom from the symptoms of dysphagia had been obtained following gastric resection carried out because of massive hemorrhage from a duodenal ulcer. On August 9, 1944, Mrs. E. K. was admitted for gastric resection, the thought being that regurgitation of acid into the lower reaches of the esophagus was the likely cause of the stricture. This operation was carried out on August 14, 1944.

At operation it was found that the patient had a congenitally short esophagus with a small paraesophageal hernia. She had been under the observation of Dr. N. Logan Leven for some time, for esophageal stricture. The question arose as to what to do for this patient. At one juncture, Dr. Leven favored resection of the esophagus with end-to-end anastomosis; moreover, the stricture was fairly high up in the esophagus. An alternative procedure would have been to have made a lateral anastomosis between the esophagus and the cardiac end of the stomach through the diaphragm from above. A third procedure which appealed to me (O.H.W.) as being perhaps most rational was to do a gastric resection as one does a gastric resection for duodenal ulcer, since it is our opinion that the esophagitis and stricture are consequent to regurgitation of free hydrochloric acid into the lower reaches of the esophagus. It had been difficult to obtain samples of free acid because of the difficulty of passing an ordinary duodenal tube through the stricture. The patient did have free acid, however, on a fasting sample as well as after the use of histamine. The quantity of acid aspirated from the stomach was large though the acidities were not unusual.

The field of operation was prepared with Novak's solution. Cyclopropane anesthesia was found to be



a b c d

Fig. 2. Case 3. a, Cork-screw stricture of lower third of esophagus. Considerable dilatation with food retention above the stricture. A portion of the stomach appears to lie above the diaphragm. Situation in April, 1944, a few months prior to operation. b, The status of the lower esophagus and residual gastric pouch 2 years after operation, August 14, 1944. c, Esophagram almost 4 years after gastric resection, May 5, 1948; there is no residual evidence of the former stricture and the cork-screw like deformity of the esophagus has disappeared. d, Normal esophagram save for persistent appearance of gastric mucosa-like folds in lower esophagus, May 5, 1948.

satisfactory. A left subcostal incision was used. At operation a fairly large cystic liver was found, the left lobe being as large as a normal sized right lobe. There were several large cysts, some of which held more than 3 ounces of fluid. Many adenomas were noted in the liver. A few small cysts were felt along the left kidney and there were many cysts in the pancreas. The right kidney seemed small and it was not certain whether or not it contained any cysts. Adhesions were noted from the omentum down to the left lower quadrant. Many diverticula were felt in the descending and pelvic colon. After discussing our findings with Dr. N. Leven and with the patient, both agreed that perhaps gastric resection was the best procedure. It was impossible to pass a duodenal tube through the stricture into the stomach prior to operation. Fluoroscopic examination revealed that the duodenal tube was in the esophagus.

The first step in the operation was to make a gastrostomy opening, through which a catheter was passed up through the esophageal stricture. Passage of the catheter was rather difficult; a small No. 22 bougie was finally pushed through. The catheter was caught above and sutured in place and a duodenal tube was pulled down. Then we used a larger bougie. Finally a fairly large bougie went through the stricture. The duodenal tube was left in the stomach, the gastrostomy opening was closed, and the resection was carried out. A group III 75 per cent resection was done, together with excision of the pylorus and antrum. A Hofmeister retrocolic type of gastrojejunal anastomosis was done with two rows of sutures, the anastomosis being made at the ligament of Treitz. The duodenum was closed with 2 rows of fine interrupted silk sutures over Petz clips. The catheter in the stomach was pushed into the proximal duodenojejunal loop. The adhesions to the left lower quadrant were all divided. There was also an area in the small intestine, perhaps in the midileum, where the gut was badly kinked by adhesions. These were cut and the bowel was made free. A tear in the surface of the bowel necessitated resuturing with fine interrupted silk. Interrupted silk sutures were used for closure of the abdominal wall. The patient stood this rather long operative ordeal quite satisfactorily and left the operating table in good condition. Despite this fact convalescence was rather slow, and the patient stayed in the hospital until the first of September, at which time she was dismissed. On September 12, she appeared in the out-patient Clinic and said that she could swallow without difficulty and was eating bread and meat. At this time she returned to her home in International Falls and was not seen in the out-patient clinic again until December 13, 1944. At this time a No. 43 French bougie went down without difficulty. The plasma proteins on December 24, 1944, were 7.1 grams per cent. She appeared to be improving continuously despite the circumstance that her appetite was none too good. On February 21, 1945, a dilator was again passed, and it was found that a No. 45 French bougie went down readily. After this there appeared to be no further need for dilatations. In February, 1946, a sound was passed and was found to go down without difficulty. In August, 1946, the patient was hospitalized for a cold and because of asthmatic bronchitis. The presence of arteriosclerotic heart disease and polycystic liver and pancreas was again reaffirmed on physical examination.

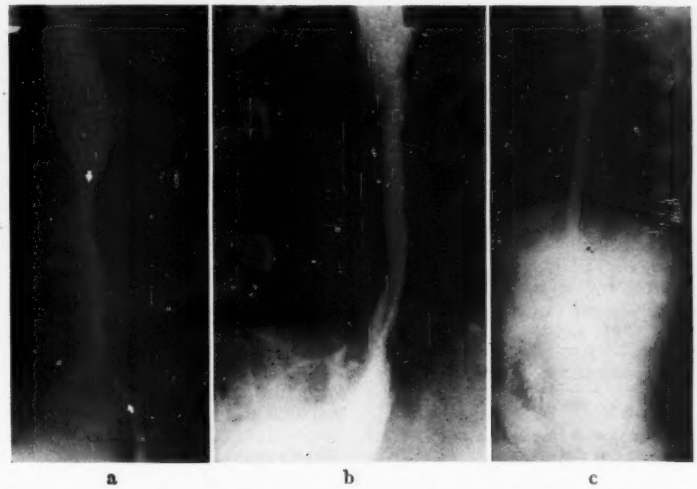


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Fig. 3. Case 4. a, Extent of esophageal stricture, November 27, 1946; patient could swallow only fluids. b, Esophagram, March 2, 1948, 8 months after gastric resection (June 27, 1947) shows disappearance of stricture and a normal esophagus. The patient can eat anything. c, Esophagram, May 18, 1948.

There has been no recurrence of hematemesis and no difficulty in swallowing since the time of the operation in August, 1944. The patient's hemoglobin has continued between 12.5 to 13 grams per cent since operation. She eats everything and seems to be getting on quite satisfactorily and has gained considerable weight. An x-ray examination of the esophagus made in September, 1944, a month after the operation, showed some narrowing of the distal third of the esophagus. An x-ray examination made 2 years later indicated that the esophagus was normal. Both proximal and distal loops of the gastrojejunal stoma fill readily. The present condition of the esophagus is shown in Figure 2, b, c, and d.

Summary. This patient had polycystic disease of the liver and pancreas, a congenitally short esophagus, and an esophageal stricture of unknown origin. There had been several episodes of hematemesis and it had been reasoned that bleeding was caused by esophageal varices followed by scar formation and esophageal stricture. Esophageal dilatation had been done approximately 100 times over a 6 year interval. Her nutrition was poor and edema and hypoproteinemia were present. Resection of the esophageal stricture had been urged but gastric resection was carried out on the thesis that the esophageal stricture was probably a manifestation of ulcer disease. The result justified the means (Fig. 2, a, b, c, and d).

CASE 4. Mr. C. G., aged 17 years, first came here in November, 1946 with a story of difficulty in swallowing (U. H. No. 772864).

The difficulty in swallowing dated back over many years, but had been getting worse and worse until the patient could no longer swallow solid foods of any kind. The hemoglobin was 16 grams per cent; the free hydrochloric acid fasting was 18 degrees with 60 degrees of free hydrochloric acid after histamine. The volume of secretion was large, being 100 cubic centimeters fasting and 88, 70, and 50 cubic centimeters respectively after three successive doses of 0.5 milligram of histamine given at 30 minute intervals. Considerable difficulty was had in dilating the patient's esophageal stricture a No. 37 French dilator causing a good deal of bleeding. An esophogram disclosed stricture of the distal third of the esophagus with considerable narrowing and some dilatation of the proximal two-thirds of the esophagus (Fig. 3, a).

Esophagoscopy was done on November 20, and disclosed stricture of the lower end of the esophagus; biopsy specimen was taken, and the histologic diagnosis was chronic inflammation. The biopsy tissue was taken 29 centimeters from the teeth. Another fragment of tissue was taken at 32 centimeters from the teeth. All these areas showed evidence of chronic inflammation (esophagitis).

Following dilatation, the patient's condition improved, and he continued to return for further dilatation at 2 week intervals. On June 27, 1947, because of the continued necessity of dilatation, a gastric resection was done (75 per cent). Inasmuch as an attempt was being made here to evaluate com-

plete extrinsic denervation of the stomach, a vagotomy and bilateral celiac ganglionectomy were carried out simultaneously. Convalescence was satisfactory and patient was dismissed from the hospital on July 3. On July 5, he returned with the picture of a psychosis and was hospitalized for a period of 13 days by the psychiatry service.

There has been considerable and continued improvement since the gastric resection in the patient's ability to swallow; moreover, when he has returned for dilatation, the tendency of the stricture to narrow down and resist stretching has not been as obvious as previously. In February, 1948, a No. 45 French sound went through without difficulty, and there seemed to be minimal evidence of any constriction. At the present time (May, 1948), the patient is eating solid foods such as bread and meat without difficulty. Moreover, the esophagram shows considerable improvement from the preoperative situation (Fig. 3, b and c). The boy appears to have made a very satisfactory functional adjustment too, and is getting on very well in school.

Summary. A boy of 17 years with a stricture of the lower half of the esophagus of unknown origin. Esophagoscopy indicated the presence of esophagitis. No ulcer could be demonstrated but the gastric acids were high. During the year prior to operation, esophageal dilatation was done, usually every 2 weeks to help maintain a flagging nutritional status. Gastric resection was followed by complete relief of the difficulty in swallowing (Fig. 3, a and b).

CASE 5. Mrs. V. G., aged 56 years (U. H. No. 667582).

Patient began coming to University Hospitals in 1938 because of painful, running ears. She was then 46 years of age. Tonsillectomy was done, and on later admissions mastoid operations were performed. In 1939 the patient underwent panhysterectomy in the gynecological department of this hospital for a uterine polyp and salpingitis. In 1942 it was noted that the patient had trouble in swallowing solid foods. When an esophagram was made in July, 1942, esophageal varices, together with a superficial gastric ulcer, were reported. These findings were confirmed by a re-examination of the esophagus in June, 1943. Gastric analysis in July, 1942, showed a large volume of gastric secretions with 66 degrees of free hydrochloric acid after histamine. Frequently during 1943 and 1944, when the stool was studied for occult blood (guaiac), blood was almost always found in the stool. A tentative diagnosis of likely esophageal stricture had been made in July, 1942, on the basis of the symptoms. The patient was put on a Sippy diet with powders because of the complaint of gastric pain and difficulty in swallowing. Foods seemed to stick in the esophagus, and there was a dull pain behind the sternum on swallowing. The patient meanwhile was observed in the psychiatric clinic, and was treated for emotional instability. By August, 1943, the patient was convinced, on the basis of her own symptoms, that she had a peptic ulcer. The patient continued to be observed in the psychiatric clinic, but by July, 1944, the physicians in the department of internal medicine in the out-patient clinic were convinced that the patient had an ulcer, together with esophageal stricture and esophageal varices.

A diagnosis of esophageal stricture and esophageal varices was established by esophagoscopic examination in September, 1947. The patient continued to complain of substernal pain on eating and persistent difficulty in swallowing. She can swallow only soft foods; meat and vegetables such as celery and carrots cannot be swallowed. A gastric analysis done with histamine on February 3, 1948, demonstrated large quantities of acid in each specimen. The highest amount of free hydrochloric acid was 100 degrees following the second half-milligram of histamine. This patient had had no dilatations of her esophagus prior to operation, but there was a history of difficulty in swallowing for approximately 6 years.

On March 15, 1948, a conventional 80 per cent gastric resection (Group III) operation was done, on the basis that the patient's digestive difficulties were probably essentially a manifestation of ulcer disease; that is, owing to the hyperchlorhydria, she probably has periodic erosions or ulcers in the stomach, as well as persistent esophagitis. The portal venous pressure was measured at the time of operation, and was found to be only 18 centimeters of saline solution. It is unlikely, therefore, that the patient has portal hypertension, or so called esophageal varices. The spleen was found to be small but, because of the conjectured presence of esophageal varices and portal hypertension prior to operation, it was thought wise to excise the spleen and to perform a biopsy upon the liver. The liver itself looked normal and proved to be so histologically. Convalescence was satisfactory and, save for some temporary diarrhea, patient got on very well. Almost from the beginning, following the operation, she swallowed considerably better. When an esophagram was made on May 4, the stream of barium went down more readily than on previous examinations.

Summary. A patient with unusual complaints who had diagnosed her own condition 5 years previously as being due to ulcer. Dysphagia also had been present over a 5 year interval. A recent gastric resection was done with relief of abdominal pain and difficulty in swallowing.

CASE 6. Mr. H. B., aged 47 years (U. H. No. 780565).

This patient has had a story of peptic ulcer since 1911. In 1940 he had a duodenal ulcer perforation which was closed surgically. Six weeks later the

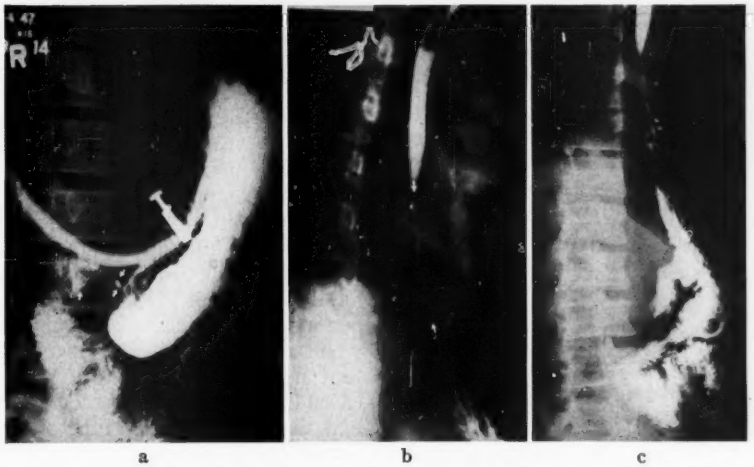

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Fig. 4. Case 6. a, When this film was made, June 4, 1947, the patient had carried a gastrostomy for several years; he could not swallow his saliva. The duodenum is deformed and there is a crater in the pyloric outlet. b, Nature and extent of stricture, March 3, 1948; a satisfactory esophagram was not obtained after esophageal dilatation. c, Esophagram made May 18, 1948, 2 months after gastric resection on March 19, 1948; the esophagram has improved as has the patient. The patient is eating solid food and is swallowing satisfactorily. The esophagus will have to be dilated periodically until it will take a No. 45 French dilator. It remains to be seen whether a normal appearing esophagus can be re-established in this patient.

obstruction of the esophagus had progressed to the extent that he could not swallow his saliva and a gastrostomy was done to permit feeding him. This was done at the Wisconsin General Hospital. Since performance of his gastrostomy he has been able to swallow liquids only periodically, usually having to expectorate saliva as well. In the summer of 1947 his physician, Dr. William Focke, of Poynette, Wisconsin, was in the clinic when the preceding case, that of C. G., was being discussed in the out-patient clinic. He had just returned a few weeks after gastric resection and reported himself as already improved. Dr. Focke said he had a patient who in many respects was very much like the case of C. G. and volunteered that he would like to send his patient here. The patient arrived in July, 1947, and an effort was made to examine the esophagus with barium, but so little barium went through, that the examination was unsatisfactory (Fig. 4, a). An attempt was made to use the esophagoscope and this also was unsatisfactory. Finally, a No. 5 ureteral catheter was passed down the esophagus into the stomach and was fished out through the gastrostomy opening. A silk thread was then brought back and anchored to the cheek. After dilatation re-examination was done with barium, and it was found that a stricture extended down from the midsegment of the thoracic esophagus to the stomach (Fig. 4, b). With many dilatations it became possible to dilate the esophagus up to about a No. 29 French. At that juncture, the patient felt much better, so he left and was not seen for several months.

Meanwhile, January, 1948, another perforation of a duodenal ulcer occurred. This time the patient was treated conservatively by suction applied to the gastrostomy tube and he appeared to get on satisfactorily. When he reported here for observation again in April, 1948, the esophagus again appeared completely occluded. The esophagus was gradually stretched up to a No. 29 French again. The patient's hemoglobin at this time was 14.3 grams; leucocytes numbered 10,500. There was a large volume of acid in every sample aspirated. Volumes of 45 to 50 cubic centimeters were present in each specimen, including the fasting specimen; the free hydrochloric acid fasting was 42 degrees and after histamine rose to 74 degrees. When the patient came here he was thin, very weak, and it was difficult for him to stand.

At the time of operation on March 19, a granuloma containing purulent material was found in the retrogastric space suggesting that the perforation in January had occurred into the lesser sac. There was a hard, thick fibrous plastic area plastered up against the transverse colon, which lent the impression in the preoperative x-ray film of a stricture there. The stomach was large. It became necessary to make an open closure of the duodenum. This was made with a single row of interrupted silk sutures. The duodenojejunal ligament was dissected down with some difficulty and 80 per cent of the stomach was re-

sected. The removed gastric segment, devoid of omentum, weighed 227 grams. A single row gastrojejunal anastomosis was made and the inlying duodenal tube was placed in the proximal duodenojejunal loop. Because of the presence of the subclinical retrogastric abscess encountered (absence of fever and leucocytosis), air vent suction drains were put into the subhepatic area on the right and behind the spleen on the left side. The patient convalesced satisfactorily, but was kept in the hospital for 2 weeks after operation to encourage better feeding. About the time the patient left the hospital it was found that a No. 29 French bougie was inserted without any difficulty. Two weeks later he returned and dilatation was accomplished up to a No. 33 French without any difficulty. A few days later it was found that a No. 35 French bougie could be passed without much difficulty; at this juncture, the patient returned home. He returned for observation again May 5, approximately 6 weeks after the operation, and said that he was eating well and had no difficulty in swallowing. At this time the esophagus was dilated to a No. 37 French with little difficulty. May 18, the patient returned at our request and it was found that a No. 37 French bougie went through without great difficulty. The patient is having very little difficulty in swallowing. Moreover, the x-ray film made at this time showed considerable improvement in the lower reaches of the stricture but still showed some evidence of spasm or narrowing at the upper end of the esophageal stricture (Fig. 4, c). The patient is eating well, however, and feels considerably improved. He eats meat, hard rolls, celery, and carrots without difficulty. Prior to the operation, it is to be remembered, the patient had difficulty in swallowing his saliva.

Summary. A patient of 47 years with a history of periodic severe difficulty from a duodenal ulcer since the age of 10. Eight years prior to admission here, surgical closure of a duodenal perforation was done followed a few months later by gastrostomy for feeding, because of the patient's inability to swallow his own saliva, occasioned by the stricture of the lower half of the esophagus. Considerable improvement following a recent gastric resection (Fig. 4, a, b, and c).[1]

ETIOLOGICAL CONSIDERATIONS

Whereas this group of cases is small, and in the instance of the last 2 patients cited, the time interval has been brief, nevertheless, it is difficult to escape the conclusion that esophagitis is a manifestation of ulcer disease.

Much has been written about the etiology of esophagitis. Vinson (1940) suggests that "nonspecific inflammation is the most common lesion of the esophagus and that the majority of benign strictures are of uncertain etiology." Vinson states that approximately 10 per cent of esophageal strictures occur as a sequel to the nausea and vomiting of pregnancy. Butt and Vinson (1936) observed that postmortem evidences of esophagitis occur in 7 per cent of individuals. Twenty per cent of that number had a history of peptic ulcer. Olsen (1948) stresses the association of abdominal disease, especially illness giving rise to vomiting as antecedents of esophagitis.

The proventriculus of the rat's stomach has the same type of epithelial covering as has the esophagus; moreover it is well known that ulcer when observed in the rat's stomach is found almost invariably in the proventriculus (2,3); similarly the histamine ulcer is provoked readily in the rat's proventriculus (1,2,3). The pig's stomach has a fairly wide ring of esophageal epithelium around the esophagogastric juncture. It is just in that area that the histamine provoked ulcer is elicited regularly (6). In other words, the squamous epithelium of the esophagus is more vulnerable to injury by the acid-peptic digestive juice than is the glandular epithelium of the stomach.

Ulcer is observed most frequently in the duodenum. And the reason probably is that, it is the sole exit through which the entire output of gastric juice escapes from the stomach. Regurgitation of the acid digestive juice into the lower reaches of the esophagus is not an uncommon circumstance as manifested by acid eructations or a sensation of substernal burning. This occurrence also has been substantiated in the fluoroscopic observations of Robins and Jankelson (1926). They observed return of the barium mixture into the lower esophagus after being swallowed in 4.6 per cent of persons examined in the prone position. If all the gastric juice left the stomach via the esophagus, esophagitis would in all probability be the most frequent of all clinical types of ulcer.

[1]In this patient dilatation is still being done at gradually lengthening intervals. He eats everything and gets on very well. None of the other patients reported herein require dilatation. One additional patient, Mr. S.N., aged 79, University Hospital No. 784106, with esophageal stricture necessitating dilatation has been subjected to gastric resection since this paper was written. He underwent operation on July 14, 1948. His substernal pain is gone and necessity for continued dilatation has disappeared.

There is a growing conviction among clinicians dealing with the upper alimentary canal that hiatus hernia interferes with effective closure of the esophagogastric juncture, thus inviting periodic regurgitation of gastric juice into the lower reaches of the esophagus. Moreover, Dey and his associates (1946) appear to have shown that the artificial creation of hiatus hernia in the dog leads to reflex shortening of the esophagus. They observed too that manipulation of the upper abdominal viscera led to contraction of the longitudinal muscle fibers of the esophagus with resultant reflex shortening. The fibrosis and contraction theory of the origin of so called thoracic stomach appears to account satisfactorily for many cases of congenitally short esophagus. In an able and critical review Smithers (1945) states that not infrequently patients in whom a diagnosis of congenitally short esophagus has been made by means of x-ray examination are found later at surgery or on postmortem examination to have the entire stomach in the abdomen. In other words, hiatus hernia may abet the ulcer diathesis not alone as it relates to the rather frequent association of gastric ulcer with the hernia but it appears also to be a forerunner of ulcer disease in the esophagus.

As one learns the natural history of a disease, he interrogates more intelligently patients having some manifestation of that disease. We feel that in this experience, we have been learning something of the natural history and a rather unexpected, though probably not bizarre, behavior of so called acid-peptic ulcer. Esophagitis may be the only manifestation of ulcer. Every patient with a duodenal or gastric ulcer should be interrogated carefully with reference to the following: periodic sensation of burning in the esophagus; acid eructations and the tasting of food long after its ingestion, substernal pain, difficulty in or pain on swallowing. And if in the roentgen-ray examination of every ulcer suspect, great care is observed to note any abnormal behavior of the lower esophagus with reference to evidence of spasm, persistent narrowing or halting of the forward progress of the barium column into the stomach, the esophagitis form of ulcer disease would be recognized and diagnosed more frequently than it is.

The gastric mucosa is the most resistant to digestion of the natural membranes with which the acid-peptic digestive mixture comes in contact. Investigators are still trying to resolve the question posed by John Hunter more than a century ago: Why doesn't the stomach digest itself? When clinicians and roentgenologists pool their information garnered in the light of the suggestion that esophagitis is a manifestation of ulcer disease, it may appear that, next to duodenal ulcer, esophagitis is the next most frequent variety of ulcer, exceeding gastric ulcer in frequency. Certainly, in all patients with obstructive duodenal ulcer and vomiting, esophagitis must be common; in any case it is in the experimental animal, a thesis which will be amplified in a subsequent publication.[1]

RATIONALE OF GASTRIC RESECTION FOR THE RELIEF OF ESOPHAGITIS AND ESOPHAGEAL STRICTURE

An effective gastric resection has two features which commend it as a suitable therapeutic agency for the relief of esophageal obstruction arising as a manifestation of ulcer disease: (1) It reduces the acidity and the digestive capacity of the juice secreted by the residual gastric pouch, a circumstance owing in part to the regurgitation of bile, pancreatic juice, and succus entericus from the short proximal duodenojejunal loop into the remaining gastric pouch. (2) Gastric resection quickens the gastric emptying time. Both these features are essential. In Case 2, reported herein, it was noted that, whereas the pyloric obstruction disappeared following performance of gastrojejunostomy, the functional manifestation of the esophagitis and esophageal stricture only came into evidence following that operation. In other words, reduction of gastric emptying time alone is not effective therapy for esophagitis or esophageal stricture due to acid-peptic ulcer of the esophagus; effective simultaneous reduction of gastric acidity is also necessary. A gastric resection which fails to reduce gastric acidity effectually cannot overcome the ulcer diathesis and will prove

[1]Since this paper was written, good experimental evidence in the dog and rat has been obtained, indicating a far greater susceptibility of the esophageal over gastric mucosa to injury by the acid-peptic digestive juice.

no more effective in the relief of esophagitis and esophageal stricture of acid-peptic origin than gastrojejunostomy.

The criteria of a satisfactory gastric resection for ulcer have been described elsewhere (7,8,14,15). Suffice it here to say that, a three-quarter gastric resection carried out on the Billroth II plan of operation with a Hofmeister end-to-side gastrojejunal anastomosis, with a short afferent duodenojejunal loop together with complete excision of the antral mucosa —these serve to constitute a satisfactory and effective gastric resection for ulcer (14).

Following gastric resection, the pool of digestive juices accumulating periodically in the residual gastric pouch loses its capacity to irritate the lower reaches of the esophagus and with periodic dilatation of the esophagus, for a few months after operation, until the normal diameter is re-established (No. 45 French), the need for continual dilatation of the esophagus disappears. Whether a normal sized esophageal lumen will return in the patient described in the protocol of Case 6, who carried a gastrostomy for 8 years during which time he could not swallow his saliva, remains to be seen. Certain it is that the less the penetration of the effects of the esophagitis into the deeper layers of the esophageal wall, the more likely is restitution of a normal esophageal lumen to be achieved.

Some speculation as to whether cardiospasm and strictures of the lower end of the esophagus owing to the swallowing of lye might be helped by gastric resection seems warranted. In any case, one of us (O.H.W.) feels that this thesis should be explored.

SUMMARY AND CONCLUSIONS

The case histories of 6 patients, presenting clinical evidences of esophagitis, esophageal stricture or both are cited, in which relief of the esophageal obstruction followed gastric resection. Three of these patients (Cases 1, 2, and 6) had an associated duodenal ulcer; one had a gastric ulcer (Case 5); one had a congenitally short esophagus and repeated hematemesis of unknown origin (Case 3). In Case 4, a boy of 17, there was a stricture of long length of the lower portion of the esophagus of unknown origin. Relief of the ulcer diathesis by a satisfactory gastric resection appears to be satisfactory treatment for esophagitis and esophageal stricture resulting therefrom; as a corollary of this experience, it is suggested that these observations constitute strong evidence suggesting that esophagitis is a manifestation of ulcer disease.

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SURGICAL TREATMENT OF PATENT DUCTUS ARTERIOSUS

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IT is only ten years since the first patent ductus arteriosus was ligated successfully by Gross (3). During this decade much has been learned about the surgical management of these cases. Although in general the operative procedure is fairly well standardized there still is considerable difference of opinion as to the best and safest method of closing the patent ductus. The medical world was greatly elated by Gross's success in getting a single ligature tied around the first ductus. Although no one wishes to shade the glory of the first success it is known today from the trial and error method that a single ligature tied snugly around a patent ductus is not sufficient to maintain occlusion in a considerable percentage of the cases. Gross (2) himself was the first to recognize this fact. He used double ligatures; tried injecting sclerosing solution between the ligatures; then added cellophane wrapping around the ligatures to stimulate snug closure by fibrosis. After treating 47 patients by these various methods and finding incomplete closure or recurrence of the fistula in 20 per cent, he abandoned methods of ligation and now routinely clamps, divides, and sutures all patent ductus.

Although this attitude is not shared by all surgeons it appears to be making headway. Jones (4) in a series of 53 ductus ligations reports aortopulmonary fistula in 2 cases, 5 early recurrences of the murmur due to incomplete occlusion and 5 late recurrences of the fistula due to the tapes partially cutting through the wall of the ductus. Jones (5) now divides and sutures all ductus and in similar series has been highly pleased with his results.

Wangensteen has concluded that the ductus should be cut and sutured. He reports division of the ductus in 33 consecutive patients without mortality or recurrence.

Blalock on the contrary believes that double ligation, one suture placed well on the pulmonary end, the other on the aortic end of the ductus, plus a transfixion suture between these ligatures, assures adequate closure. If there is any uncertainty about complete closure he adds a ligature of umbilical tape.

Shapiro in an analysis of 643 patients operated upon by 46 surgeons reports a mortality rate of 4.9 per cent and a recanalization rate of 8.7 per cent. He concluded from a study of 172 cases with uninfected ductus severed and sutured without mortality or recanalization that ligation of the patent ductus is now obsolete.

My attitude toward the surgical treatment of patent ductus arteriosus is colored by my own experience. To divide and suture a ductus is not a simple operation and should not be undertaken until one has familiarized himself on animals with the handling and suturing of large vessels. For trained surgeons to whom the opportunity of doing practice vascular surgery on animals has not been available, I believe that proper ligation of a patent ductus is a safer procedure. Furthermore, any surgeon is wise to begin his operative work in this field on children because the operation is much easier in a child than in an adult.

At the Children's Memorial Hospital in Chicago the management of patent ductus has gone through two phases of surgical approach and surgical management.

In the first 12 cases an anterior submammary incision was made (Fig. 1). The chest was opened through the left third interspace and sufficient exposure was obtained by cutting the second and third ribs at the sternal margin. In children the ribs are pliable and the upper flap of ribs and attached muscle can easily be held aside with a claw retractor. A rib spreader may well be used but unless one is constantly on guard the lower flange is apt to rest against the heart and influence its rhythm unfavorably. In the next 28 cases a posterolateral approach has been used (Fig. 2),

From the Children's Memorial Hospital, Chicago, Illinois.

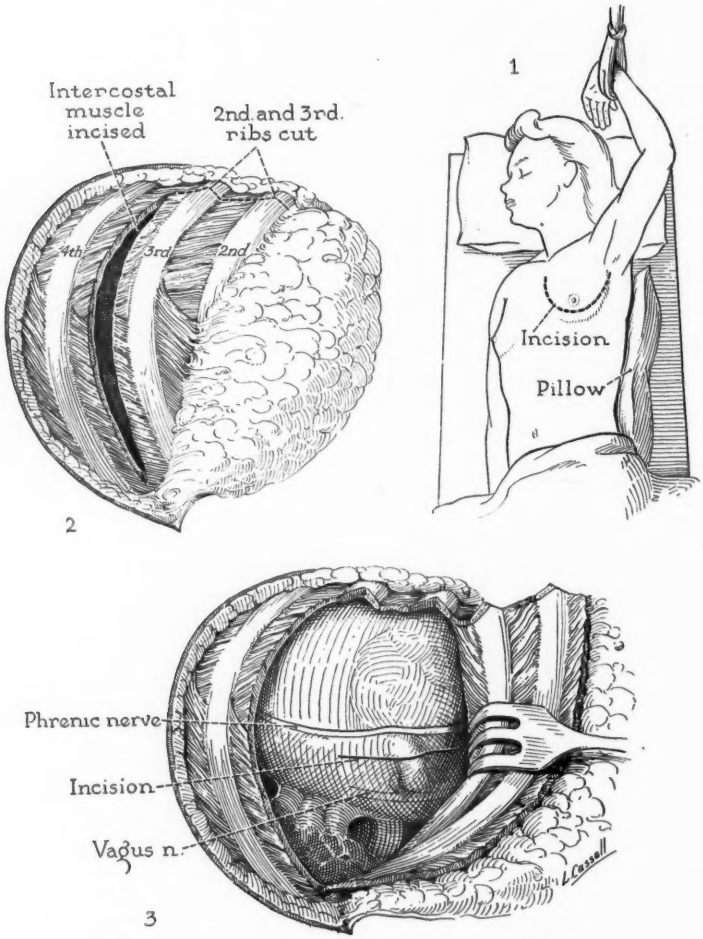


Fig. 1. Anterolateral approach to patent ductus arteriosus.

and the chest entered through the fourth left interspace. This incision is easier to make, gives better exposure, is easier to close, and leaves a less conspicuous scar.

The first 22 consecutive patent ductus were ligated as illustrated in Figure 3. There was no mortality and to our knowledge there has been no recanalization. In spite of the latter fact, which may well be coincidental and judged by the experience of others will undoubtedly change, and because of the fact that it is not the best surgery to bury large amounts of nonabsorbable foreign material on large vessels and against a bronchus, investigation was undertaken for the development of a ductus clamp which would be effective, nontraumatic, and safe. It appears that such a clamp has been developed (Fig. 4). The basic feature of this clamp, previously described (6), is embodied in a row of very fine teeth in the apposing jaws. The principle of the Indian fakir who walks on spikes has been applied to a clamp. He walks on spikes without injury to his feet only because he steps on many points at one time. Likewise, a clamp with fine teeth can be applied to a vessel without

injury only because the many teeth distribute the pressure widely. The serrated portion of the jaws of the clamp, 21 millimeters long, and 1 millimeter wide is hollow ground so that at the base of the teeth it is about 0.5 millimeter wide. There are 40 teeth to the inch (16 to 1 cm.). Each tooth is about 1 millimeter long. The hub of the clamp is so constructed that the jaws can be closed to a point of complete apposition but not interdigitation of the teeth. When closed the teeth embed themselves in the adventitia and will not slip. Experimental studies on dogs indicate that any of the large vessels may be adequately occluded without tissue injury. It is important that these clamps be made of good, well-tempered stainless steel.

OPERATION

The operative technique as used on 18 consecutive patients is as follows (Fig. 5): Intratracheal anesthesia is used. A cannula or large needle is placed in the right saphenous vein at the ankle for the administration of fluids or blood if necessary. We routinely have in the operating room 500 cubic centimeters of blood. The child is laid on its right side and a curved incision is made beneath the left scapula. The chest is opened through the left fourth interspace. In children below 12 years rib resection is rarely necessary.

The lung is collapsed, covered with a moist pad, and held out of the way with a two-bladed retractor made of flexible monel metal. At this point it is important for the anesthetist to stop spontaneous respiration by breaking the Herring-Brauer reflex and breathing for the patient by intermittent pressure on the anesthetic bag. By so doing, the surgeon is greatly aided because movement of the diaphragm is stopped and with it the up-and-down motion of structures in the chest.

The phrenic nerve is easily identified and about 1 centimeter posterior to and parallel with it the mediastinum is opened. The ductus is readily located by palpating the point of maximum thrill. The vagus nerve and its recurrent laryngeal branch coursing around the ductus is next identified. A number of lymph nodes usually lying about the ductus are wisely dissected out and removed because they interfere with clear visualization.

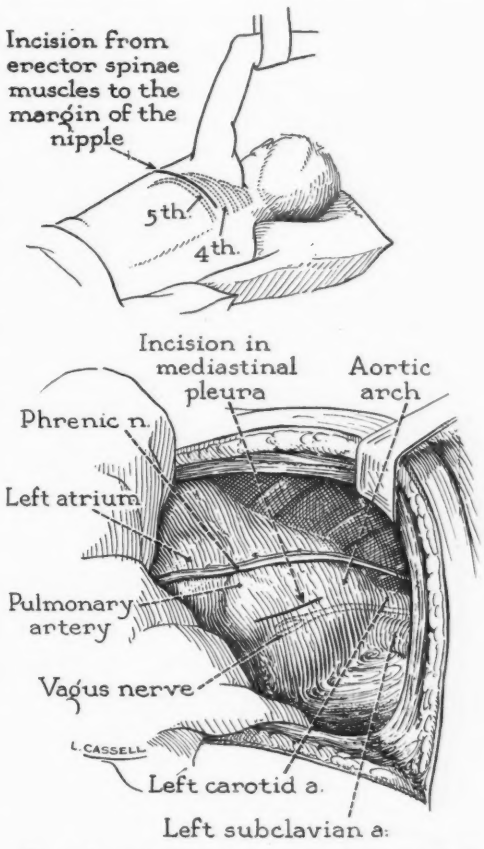


Fig. 2. Posterolateral incision and surgical approach to patent ductus arteriosus.

Careful, painstaking, unhurried, and adequate dissection of the ductus from its surrounding structures is the important step in this operation. A lappet of pericardial sac usually overlies the ductus and must be completely separated from it. The proper line of cleavage for this dissection is through the adventitial layer. (In one patient I failed to dissect the pericardium completely from the pulmonary end of the ductus. When the clamp was applied the teeth held in the pericardium but one side of the cut ductus tended to retract and made suturing difficult.) The posterior portion of the ductus must be dissected from the bronchus blindly. Again, if

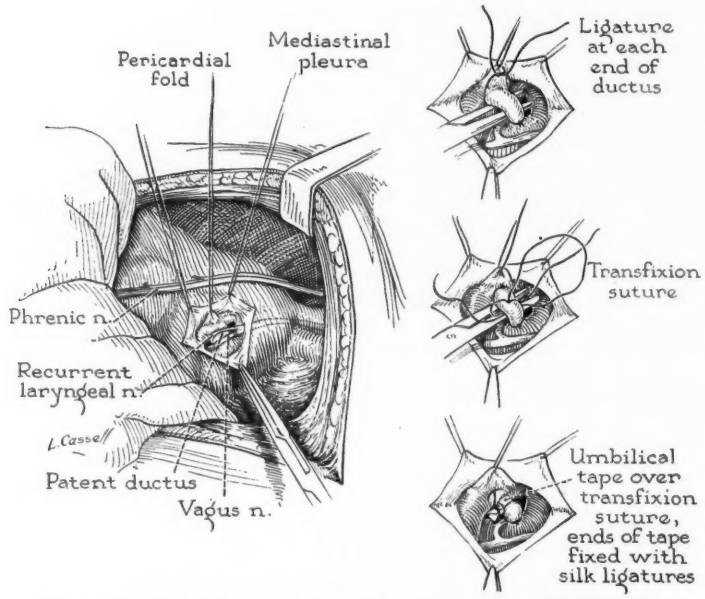


Fig. 3. Exposure of ductus and method of obliteration by multiple ligatures.

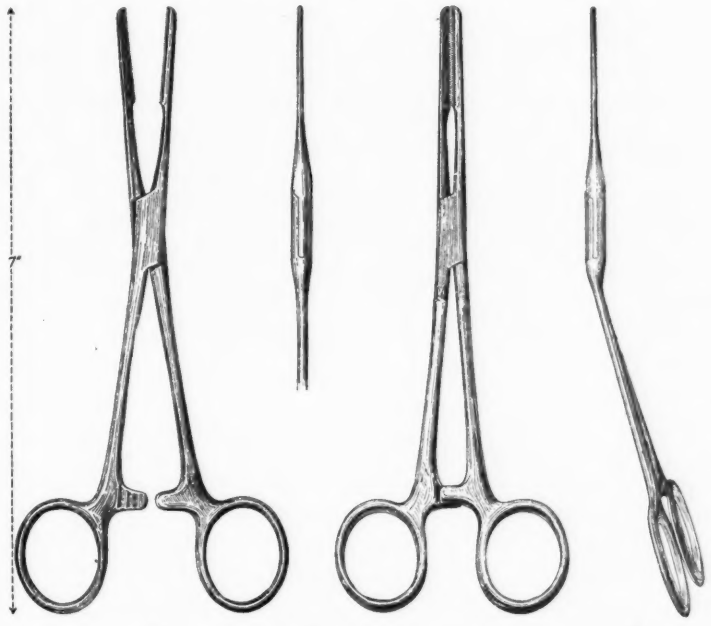
the line of cleavage through the adventitia is followed this step will be simplified.

After a curved cystic duct forceps has found its way beneath the ductus a strip of umbilical tape is drawn through to elevate it while the dissection is completed, first on the aortic side and then on the pulmonary side. The tape is then removed and while the ductus is elevated with a curved forceps the angled clamp is applied to the aortic end of the ductus. If the ductus is short the clamp is crowded as far as possible toward the aorta. While slight traction is being made on this clamp to elevate the ductus, the second clamp is applied on the pulmonary end.

With a long straight scissors, designed by Dr. Sidney Smith, a small cut is made in the ductus. If no bleeding occurs (none has) it is completely severed midway between the clamps. The stumps distal to each clamp are usually 2 to 3 millimeters long.

The aortic stump is sutured first. No. 5-0 Deknatel silk swaged on a curved No. 9 atraumatic needle has been found very satisfactory. Until recently each stump was closed with a double row of a continuous over-and-over suture. Because occasionally there was some bleeding between the stitches we now use a continuous mattress suture all the way across the end of the ductus, then return with the same suture in an over-and-over manner at a slightly different level and tie the ends at the point where the sewing was begun. The clamp is partially released. If there is any bleeding the clamp is closed and an extra stitch or two is put in where necessary. A strip of gelfoam is then laid over the suture line and the aorta is retracted gently with a malleable retractor while the pulmonary end of the ductus is sutured in a similar manner.

The mediastinal pleura is closed with a few interrupted silk stitches. Just before the last stitch is drawn up, penicillin, 100,000 units in 1 cubic centimeter of water, is sprayed over the ends of the sutured ductus. The lung is completely re-expanded by the anesthetist. A No. 16 or No. 18 F. de Pezzer catheter with all but the flange removed is inserted from the inside out through a small puncture wound in the sixth interspace. The ribs are apposed with two catgut sutures about adjoining ribs. To avoid pressure on the intercostal nerve the



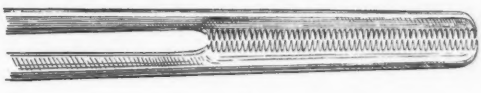


Fig. 4. a and b, the straight clamp used on the pulmonary end of the ductus; c and d, the angled clamp for the aortic end of the ductus; e, a much enlarged drawing of the teeth of the clamp.

suture about the lower rib is put in subperiosteally. The muscles are closed with a running catgut suture and the skin is closed with silk.

While the anesthetist makes pressure on the anesthetic bag to force all the air from the chest the drainage catheter is clamped. After the patient is returned to bed the catheter is attached to a water seal. On the second or third postoperative day the catheter is removed. Penicillin, 30,000 units every 3 hours, is given intramuscularly for 5 to 7 days after operation, routinely.

The results in the second group of 18 patients in whom the ductus was severed and sutured have been completely satisfactory. There were no deaths and no postoperative complications.

DISCUSSION

Mortality and morbidity statistics will always be lower in children because cardiac reserve has not been depleted and because in children the ductus is longer, more elastic, and more easily freed from surrounding structures.

The diagnosis of patent ductus in this series of cases was made by Dr. Stanley Gibson and confirmed at operation in every case.

The ages of these patients varied from 2 to 13 years. The most suitable age for operation is between the third and seventh year.

Postoperative complications although not troublesome, occurred in the first group of patients. One child, aged 4, had a severe postoperative tracheobronchitis and a collapse of the left lung. Both cleared spontaneously. Temporary paralysis of the recurrent laryngeal nerve occurred in two patients—the second and fourth child operated upon. Both of the injuries occurred because during dissection the proper line of cleavage was not followed and the ductus was accidentally

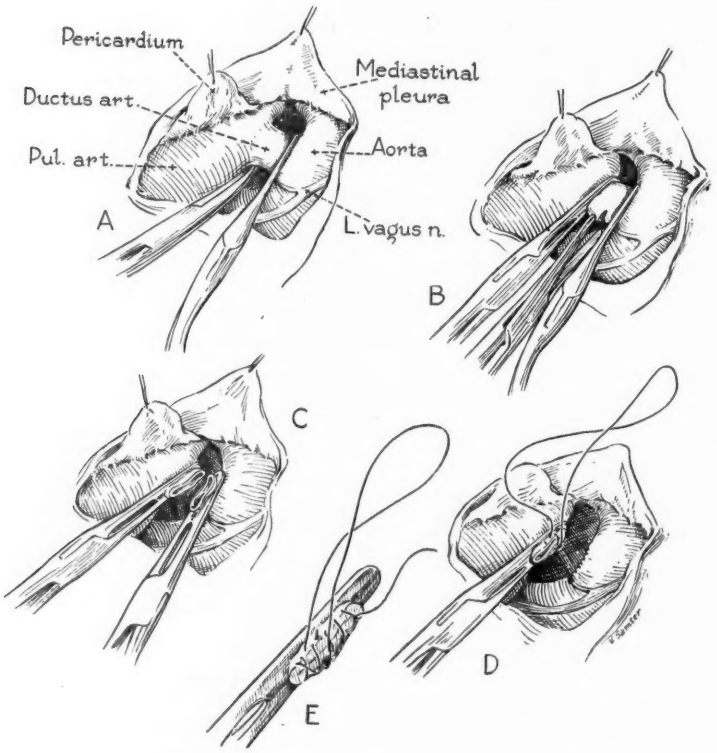


Fig. 5. Technique of division and suture of patent ductus arteriosus.

opened. In the rather panicky application of clamps the nerves were pinched.

We have had no reason to regret drainage of chest after operation; no case of empyema or infection around the tube has developed.

The outside diameter of the ductus was usually about 6 to 8 millimeters; some were as small as 4 millimeters others as large as 12 millimeters. One child, aged 13, weighing 52 pounds, with a much enlarged heart on the verge of failure had a ductus which actually measured 18 millimeters in diameter. During the first two months after operation she gained 22 pounds.

CONCLUSIONS

1. Surgical obliteration of a patent ductus arteriosus has become an accepted procedure.

2. Whether the ductus should be ligated or divided is discussed.

3. Although 22 patent ductus have been successfully ligated it is believed that the preferable operation is division and suture.

4. Employing ductus clamps which utilize a new principle of safety 18 consecutive ductus have been divided and sutured.

5. There has been no mortality in either group.

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ANATOMIC VARIATIONS OF THE EXTRAHEPATIC BILIARY SYSTEM AS SEEN BY CHOLANGIOGRAPHIC STUDIES

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CONTRARY to accepted opinions the gall bladder and the bile ducts present many variations in configuration and anatomical positions. Textbooks on anatomy fail to describe these anatomical aberrations, thereby creating the impression that the extrahepatic biliary system follows an immutable structural pattern. Such inferences are not correct.

The fact that the gall bladder may be imbedded in, adherent to, or suspended from the ventral surface of the liver invites many positional variations. For example, in short stocky people, the liver may be small and situated high under the costal margin, thereby making the gall bladder a resident of the upper abdominal cavity. On the other hand, in the tall, ptotic individual, the lower surface of the liver may extend below the crest of the ilium, thus placing the gall bladder in the recesses of the lower abdomen. The bile ducts, which pass down along the free edge of the hepatoduodenal ligament, are not rigidly anchored and therefore have no constant position.

Our studies indicate that the bizarre structural pattern of the extrahepatic biliary system occurs with such regularity as to present a most annoying problem. How can the surgeon avoid injuring the misplaced ducts, perform plastic and reconstructive operations, or even locate the elusive intraductal stones unless he is conversant with these variables? Likewise, the radiologist is unable to interpret contrast cholangiograms correctly unless he is familiar with these mutations.

The material herein presented represents roentgenographic visualizations of the extrahepatic biliary system, obtained during and after operations on the biliary tract. These contrast cholangiograms were obtained by injecting radiopaque solutions, such as diodrast, into the gall bladder and bile ducts and then exposing an x-ray film. The resulting cholangiogram gave an excellent roentgenographic pattern of the entire extrahepatic biliary system. Exact anatomical patterns of size, shape, and position of each segment of the biliary system were obtained.

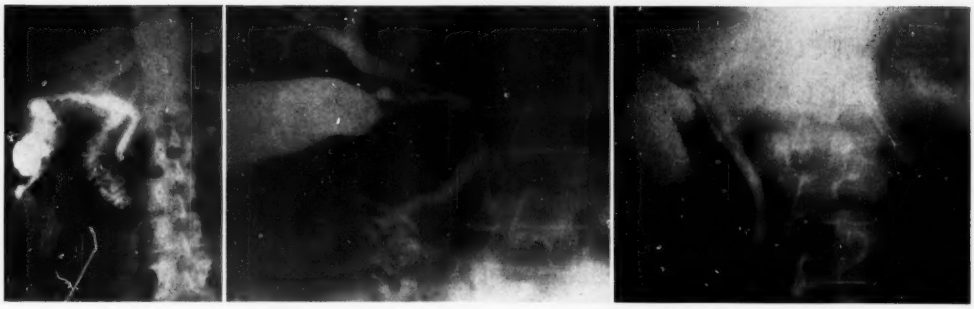
A uniform technique was employed in all operative cholangiograms in order to minimize roentgenographic distortions. The Lysholm grid was placed directly under the back of the patient in such a manner that it included the entire upper abdominal cavity. The x-ray tube stood 25 inches above the casette and was centered on a point one inch above and one inch to the right of the umbilicus. The rays were directed perpendicularly to the casette. All films were taken during a period of sustained apnea, in order to minimize distortions resulting from motion of the thoracic cage. As all anesthetics were administered by endotracheal intubation it was possible to distend the lungs by positive pressure and thus suspend respiration until the cholangiogram was taken.

OBSERVATIONS

A study of the cholangiograms reveal an amazing variation in size, shape, configuration, and position of all components of the extrahepatic biliary system. Representative cases are presented.

Gall bladder. The visualized gall bladders represented in Figures 1 to 8 indicate that this organ may occupy any position in the right side of the abdominal cavity. It may rest against the lateral abdominal wall (Fig. 1),

From the Departments of Surgery and Radiology, the University of Utah Medical School and the Latter Day Saints Hospital.



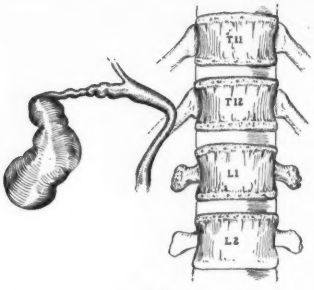


Fig. 1.

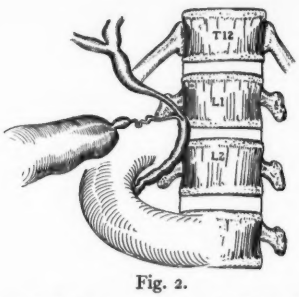


Fig. 2.

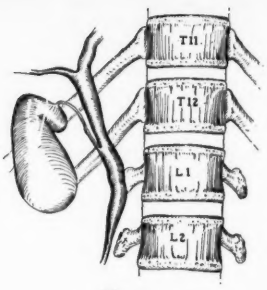


Fig. 3.

Fig. 1. Postoperative cholangiogram obtained by injecting a persisting external biliary fistula of 3 years' duration with diodrast. The stone laden gall bladder occupies a transverse position lying up under the twelfth rib. Apparently the gall stones produced an intermittent obstruction of the cystic duct, thereby forcing bile and mucus out through external orifice of the fistula. The long, sacculated cystic duct passes medially in a transverse plane to unite with the horizontally placed common hepatic duct. The choledochus first passes medially and then curves laterally so as to course downward and to the right, thus forming a partial arc. Note that the papilla of vater is placed lateral to the lower border of the body of the second lumbar vertebra.

Fig. 2. Operative cholangiogram. The distended gall bladder occupies a true transverse position. The tortuous, sacculated cystic duct courses medially in a transverse plane to unite with the horizontally situated common hepatic duct. The cystic duct forms a junction with the anterior surface of the common hepatic duct rather than assuming the usual lateral position. Again the common bile duct assumes a half crescent shape as it passes downward and to the right of the bodies of the first, second, and third lumbar vertebrae.

Fig. 3. Operative cholangiogram. This gall bladder assumes a vertical position as it is suspended from the ventral surface of the liver. Note the presence of Hartmann's pouch, which is not seen in Figures 1 and 2. The cystic duct descends along the lateral margin of the common hepatic duct for a considerable distance before effecting its union. The choledochus assumes what is considered a normal position, passing downward and slightly medially. The papilla of Vater is lateral to lower border of second lumbar vertebra.

or it may be surperimposed over the body of the third lumbar vertebra (Fig. 8). Occasionally it assumes a horizontal position (Fig. 2), or it may hang downward so its fundus extends into the upper pelvic cavity as in Figure 5. Again the gall bladder can have an "S" shaped configuration (Fig. 7), or resemble a small ovoid light globe (Fig. 4). It may present a smooth pearshaped outline (Fig. 6), or exhibit an irregular sacculated effect as in Figure 3. Rockwood encountered a gall bladder lying external to the muscles of the abdominal wall, with a cystic duct which insinuated its way between the fibers of the muscles, and traversed the fascial planes and peritoneum until it reached the common hepatic bile duct. This evidence proves that the gall bladder does present many variations as to size, location, and shape.

Cystic duct. The positions of the cystic duct quite naturally varies with those of the gall bladder which it drains. The cystic duct may appear as a direct continuation of the gall bladder (Fig. 1), or it may appear as a sacculated tube lying in a horizontal plane (Fig. 2). Occasionally it courses upward, turns

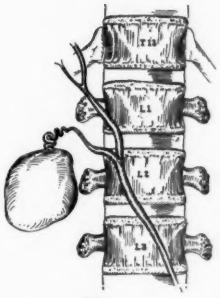


Fig. 4.

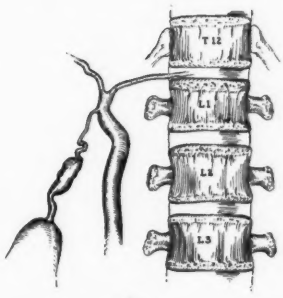


Fig. 5.

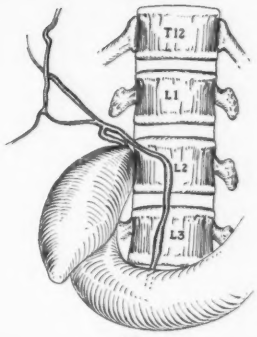


Fig. 6.

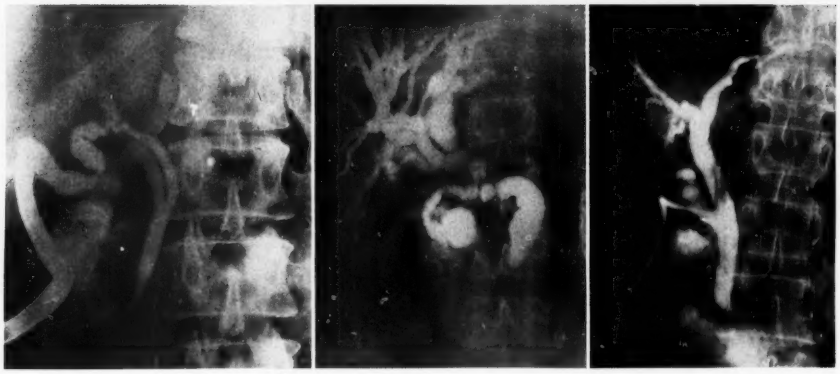
Fig. 4. Operative cholangiogram. The small ovoid gall bladder hangs suspended from the ventral surface of the liver but it does not possess a pouch of Hartmann. The cystic duct passes upward, arches medially and then passes horizontally to unite with common hepatic duct, directly over the body of the second lumbar vertebra. The slender common duct passes diagonally downward and to the left as it descends over the bodies of the third and fourth lumbar vertebrae. Note that the papilla of Vater is practically in the midline, while in the former cholangiograms it lies lateral to the vertebral column.

Fig. 5. Operative cholangiogram. This vertically placed gall bladder represents the "ptotic type," with its fundus reaching below the crest of the ilium. Note how the long, sacculated cystic duct passes directly upward to join the common hepatic duct. The dilated choledochus is vertically placed but occupies a lateral position which is in striking contrast to the medially placed ducts in Figure 4. Note the calculus obstructing the ampulla of vater which accounts for the dilatation of the choledochus.

Fig. 6. Operative cholangiogram. This pear-shaped gall bladder is nestled against the bodies of the second and third lumbar vertebrae, and is practically contiguous with the proximal segment of the common bile duct. Note how the cystic duct passes in a horizontal direction toward the right lateral abdominal wall, then turns on itself and passes medially to meet the transverse common hepatic duct over the body of the second lumbar vertebra. The choledochus is pushed over to the midline so it lies over the midportion of the lumbar vertebra, a distinct variable from Figure 5.

medialward and then passes downward thus forming a semicircle (Fig. 4). At times the cystic duct lies contiguous to the common hepatic duct as the two descend downward between the leaves of the hepatoduodenal ligament for a distance of 3 inches before the two ducts unite (Fig. 3). At other times the entire cystic duct courses upward in a perpendicular direction (Fig. 5). In Figure 8 the cystic duct encircles the gall bladder, the latter being mesial and ventral to its evacuating duct.

The manner in which the cystic duct forms its junction with the common hepatic duct is very interesting. As a rule the cystic duct empties into the right lateral wall of the common hepatic bile duct (Fig. 1). It may, however, open into the anterior surface of the common hepatic duct (Fig. 2), or even more rarely into the left mesial wall of the



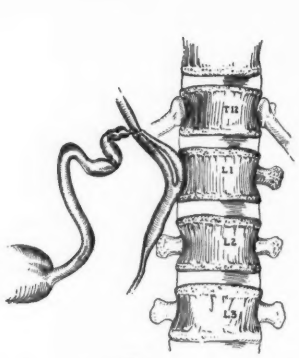
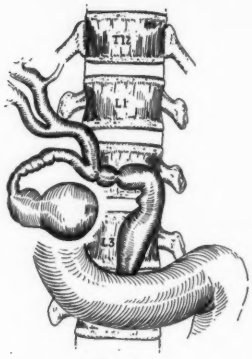


Fig. 7.



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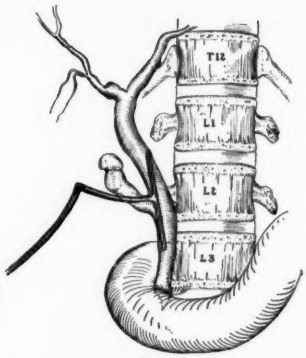


Fig. 9.

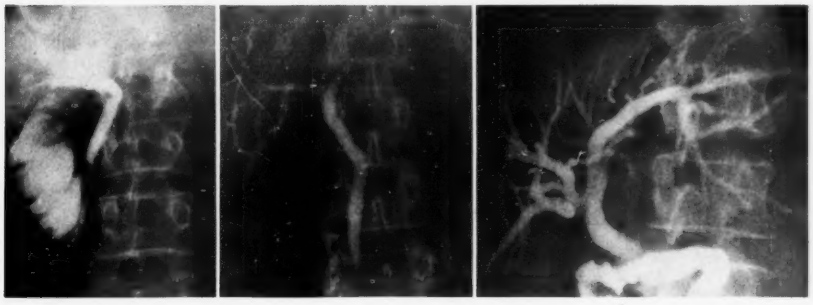
Fig. 7. Operative cholangiogram. The "S" shaped gall bladder is transversally placed so it lies under the eleventh rib. The long, tortuous, dilated cystic duct assumes an unusual pattern. Note how it first passes upward, then arches medially so it lies anterior to the common hepatic bile duct. It then passes downward along the medial margin of the common hepatic duct, so the two are contiguous. The choledochus has a normal position with the papilla of Vater being lateral to the lower border of the second lumbar vertebra.

Fig. 8. Operative cholangiogram. The fundus of the contracted gall bladder lies in the midline, directly over the body of the third lumbar vertebra, while the cystic duct passes first to the right, then upward, and finally to the left to join the common hepatic duct over the midportion of second lumbar vertebra. The first portion of the dilated choledochus passes transversely across the body of the second lumbar vertebra, angulates and descends over the left side of the third lumbar vertebra. The ampulla of Vater was not visualized because it contained several stones which had displaced the contrast media.

Fig. 9. This postoperative cholangiogram depicts the normal position of the bile ducts. The common hepatic bile duct descends in a vertical direction just lateral to the bodies of the first and second lumbar vertebrae. The choledochus which is formed by a juncture of the cystic and common hepatic ducts at the level of the lower margin of the second lumbar vertebra, and from here it descends in a vertical plane. The ampulla of Vater was not visualized because it contained several stones which had been overlooked at the primary operation, but despite this obstruction much of the diodrast had escaped into the duodenum. Note the long, dilated stump of the cystic duct resulting from the hydrostatic pressure incident to the ampullary obstruction.

common hepatic duct as the two form the choledochus (Fig. 7).

Common hepatic bile duct. The common hepatic duct is usually formed by a union of the right and left hepatic ducts at the level of twelfth thoracic vertebra. It is generally believed that the newly formed common hepatic duct descends along the free edge of the hepatoduodenal ligament in a vertical plane (Fig. 3), but it was interesting to observe the frequency with which it assumes a horizontal position (Figs. 1, 2, 8, 12, 15). This horizontal pattern must be kept in mind when exploring this duct for floating calculi.



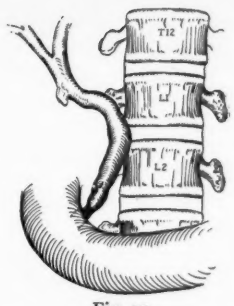
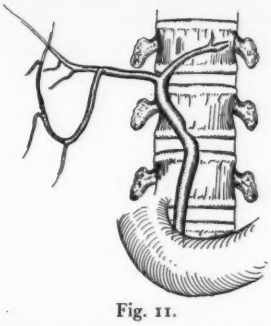


Fig. 10.



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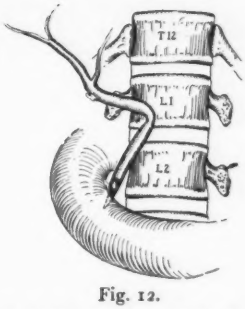


Fig. 12.

Fig. 10. Operative cholangiogram. The dilated common duct assumes a "half crescent shape" rather than descending in a vertical place. The proximal segment is in a horizontal position, while the distal portion passes downward and to the right. This produces an acute angulation. The shadow in the ampullary area represents an impacted stone.

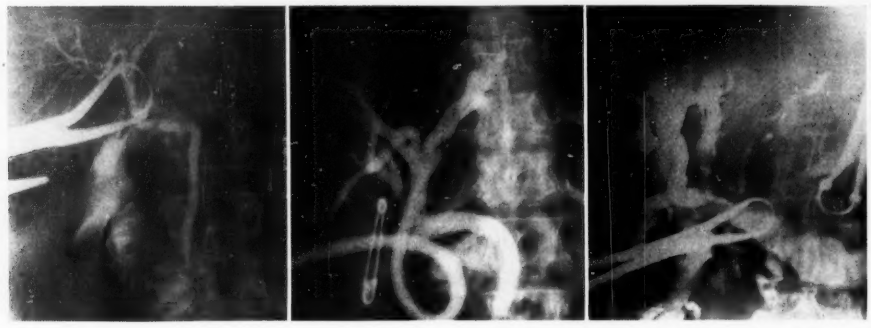
Fig. 11. Operative cholangiogram. The common hepatic bile duct and the choledochus traverse a vertical plane as they pass downward over the midportion of the second, third, and fourth lumbar vertebræ. The papilla of vater does not pass to the right of the vertebral column before penetrating the duodenal wall. The entire extrahepatic biliary system thus occupies a midline position.

Fig. 12. Operativecholangiogram. The common hepatic duct assumes a transverse position as it passes medially to unite with the cystic duct over the midportion of the second lumbar vertebra. The proximal segment of the choledochus is in a horizontal plane, it then angulates and passes downward to the right. Angulation of the choledochus is of common occurrence.

Choledochus. The common bile duct has no constant pattern or position. The textbook description is depicted by Figure 5, wherein the choledochus descends along the lateral margin of the hepatoduodenal ligament, lying lateral to the right margins of the bodies of the second and third lumbar vertebra. As the retroduodenal segment of the choledochus penetrates the duodenal wall it curves laterally, so the papilla of Vater is more laterally placed than any other portion of the duct (Figs. 1, 3, 7, 10, 12). However, the choledochus may pass downward in a vertical plane (Figs. 6, 11), or it may travel in a left oblique direction (Fig. 4.), or it can transcribe a "zigzag" pattern as in Figure 13. In many instances the common bile duct lies directly over the bodies of the second and third lumbar vertebra, rather than being lateral to them. Again the choledochus may form a definite semicircle (Figs. 2, 10, 13) or it may lie in a horizontal plane (Fig. 15). One is amazed at the inconstant anatomical patterns presented by the common bile duct and this may account for some of the difficulties which the surgeon encounters when he attempts to extirpate intraductal stones.

GENERAL CONSIDERATIONS

It is apparent that the surgeon must be conversant with the anatomical variants of the extrahepatic biliary system if surgical treatment is to be effective. True, the gall bladder can usually be found by exposing the



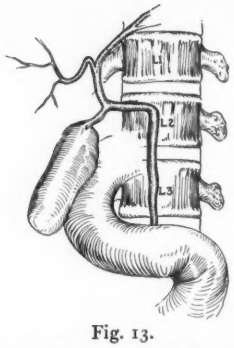


Fig. 13.

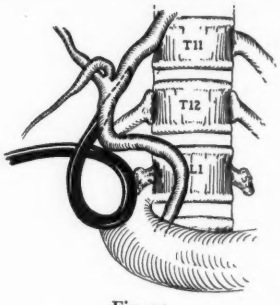


Fig. 14.

Fig. 15.

Fig. 13. Operative cholangiogram. The common hepatic duct lies lateral to the body of the first lumbar vertebra, where it unites with the cystic duct. The proximal portion of the choledochus is horizontally placed, it then forms a right angle turn and passes downward in the midline. The common hepatic and choledochus thus produce a "stair step pattern."

Fig. 14. Postoperative cholangiogram. The common hepatic duct lies high in the abdominal cavity lateral to the right margin of the twelfth thoracic vertebra. The choledochus forms a semicircle as it arches medially, and then passes downward to the lateral border of the third lumbar vertebra.

Fig. 15. Operative cholangiogram. This patient had a large carcinoma of the head of the pancreas which was obstructing the ampulla of vater. Note the dilated common hepatic duct which lies in a horizontal plane. The cystic duct unites with the posterior surface of the common hepatic duct. The dilated common bile duct has been pushed upward and medially so it traverses a direct horizontal plane. The ampulla of Vater is displaced to the left of the third lumbar vertebra. Appreciation of these transverse positions is essential for correct surgical approach.

ventral surface of the right lobe of the liver, but in Figures 5 and 8, the gall bladders have no contact with the liver except by means of the common hepatic duct and its two hepatic tributaries. In one instance we found an elusive gall bladder in the lesser omental cavity. Indeed it was so well hidden that the surgical notes of the two earlier operations stated that the "gall bladder was missing." When dealing with an acute suppurative cholecystitis the problem of locating the gall bladder is much more complicated. If the inflammatory process has spread to the stomach, duodenum, pancreas, colon, and omentum one may experience great difficulty in isolating the "enmeshed but distended gall bladder." For example, if the gall bladder seen in Figure 8 should become acutely inflamed it might readily be mistaken for a pancreatic, mesenteric or duodenal cyst, because of its unusual location.

In a recent publication we have emphasized the manner in which the cystic duct can and does complicate surgery of the biliary system. When the cystic duct is short and effects an immediate fusion with the common hepatic duct both of these structures may be included by the cystic duct ligature. When the cystic

duct descends along the side of the common hepatic duct the surgeon may leave a long residual stump which eventually becomes dilated and produces the "reformed gall bladders of Beye" (Fig. 9). Should the cystic duct cross over the common hepatic duct and unite with its medial wall, then the laterally placed common hepatic duct could be mistaken for the cystic duct, ligated, and thus produce an obstructive jaundice (Fig. 7). These errors of commission can best be minimized by obtaining cholangiographic visualizations of the biliary system prior to clamping or ligating any of the ducts.

The choledochus has always been a surgical puzzle. Even when the surgeon locates the duct he is never certain that all of the offending calculi have been removed. The large number of patients requiring secondary operations on the biliary tract provides adequate proof that such a problem exists. The angulations, unusual configurations and the anatomical displacements of the common bile duct merely increases the inefficiency of intraductal explorations. One has but to study Figures 6, 8, 13, 14, 15 to appreciate some of the problems which confront the surgeons during these intraductal explorations.

When the common bile duct presents any unusual configuration or position it is an easy matter to force an exploring probe through the duct wall thus forming an internal biliary fistula. In observing numerous operators probing the common bile duct they all attempt to pass the probe downward in a vertical plane, believing this to be the normal position of the choledochus. When rigid instruments are forced along the lumen of the angulated choledochus traumatic perforations may ensue. Such errors can be completely eliminated by first determining the exact configuration of the bile ducts by means of cholangiographic studies.

During the past 13 years it has been our practice to obtain a visualizing cholangiogram as soon as the abdomen is opened, before any portion of the extrahepatic biliary tract has been disturbed. This can be accomplished by introducing a solution of diodrast into the gall bladder or the bile ducts and then taking an x-ray film. Within a period of 10 minutes the radiologist presents the surgeon with an accurate roentgenographic pattern of all components of the biliary system. These visual patterns have been very helpful in determining the type of corrective operation to be employed.

SUMMARY

1. Roentgenographic studies of the extrahepatic biliary systems of patients during and after operations have been made. This was accomplished by injecting a solution of diodrast into the gall bladder or bile ducts and then taking an x-ray film. These cholangiographic studies present accurate roentgenographic outlines, denoting the size, shape, configuration and anatomical position of each segment of the extrahepatic biliary system.

2. The gall bladder was found to occupy almost every position in the right side of the abdominal cavity. It was seen to lie under the eleventh rib, to be contiguous to the right lateral abdominal parietes, to be pushed so far medially as to superimpose the body of the third lumbar vertebra, and even to hang down into the recesses of the pelvic cavity.

3. The cystic duct was seen to pass medially in a horizontal plane, to descend vertically downward toward the lateral border of the third lumbar vertebra, to ascend perpendicularly from a gall bladder situated in the pelvic cavity, or to describe a tortuous course as it passes mesially to unite with the common hepatic bile duct. It was noted that the cystic duct united with the right lateral wall, the anterior surface, or even the left mesial wall of the common hepatic bile duct. There seemed to be little uniformity in pattern.

4. The common hepatic duct was usually noted as passing downward and medially to unite with the cystic duct. At times, however, it occupied a horizontal position.

5. The choledochus presented no uniform pattern. In some instances it descended in a vertical plane, again it was found to lie in a transverse position. Sometimes it appeared as a vertical tube, then again it would present a "zig-zag" outline, or even be so distorted as resemble the letter "S." Occasionally it presented a tortuous contour, even transcribing semicircular forms. At times the chole-

dochus was found to lie 2 inches lateral to the bodies of the second and third vertebra or to be directly superimposed over these structures. In one instance it occupied a transverse plane throughout its entire length, crossing to the left side of the vertebral column.

6. Recognition of anatomical variations of the extrahepatic biliary system is essential before remedial procedures can be employed. Injuries to the bile ducts can best be avoided by knowing their exact position before they are molested. This can be accomplished with operative cholangiography.

A RATIONALE FOR THE SURGICAL TREATMENT OF DUODENAL ULCER

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THE most rational explanation for the maintenance of chronic peptic ulcer in the human is the inability of the mucosa to withstand the proteolytic activity of the gastric contents. This may be due to (a) lowered resistance of the mucosa, including alteration of its protective mucous secretion, (b) increased proteolytic activity of the secretion, including reduction of the diluting and neutralizing influence of saliva and upper intestinal contents, or (c) a combination of these factors.

While acute peptic ulcer may be initiated in the experimental animal by numerous etiological factors, chronic ulcer is maintained only by a persistent reduction of the mucosal resistance-proteolytic activity ratio (4, 21). The formation of a chronic jejunal ulcer following the Mann-Williamson operation depends upon shunting of the strongly alkaline biliary, pancreatic, and other upper intestinal juices away from the gastric outlet, so that undiluted, unneutralized gastric contents impinge on the jejunal mucosa (19, 20). The production of peptic ulcer by histamine or caffeine implantation depends upon increasing the proteolytic digestion (5, 16, 24, 27, 37). Lysozyme, by depriving the mucosa of its protective mucous coating, may predispose to ulceration (22, 23). Vascular or other nutritional factors may reduce mucosal resistance (4, 30).

The chief mechanisms for the stimulation of gastric secretion are the humoral and vagal phases (Fig. 1). The humoral phase is normally initiated by the action of food on the pyloric part of the stomach or in the intestine (6, 14, 33, 34). It may also be brought about by alcohol, caffeine, or histamine (8, 24, 26, 27, 28). The vagal (cephalic) phase depends upon the transmission down the vagus nerve of impulses that originate in the higher centers because of normal (thought, smell, or taste of food) or abnormal (emotional) psychic phenomena (3, 9, 36). The vagal phase may also be increased by organic brain lesions or hypoglycemic stimulation of the nerve center (7, 15). An important factor in gastric secretion is the fact that the gastric juice secreted by the humoral route is high in acid but low in pepsin while the secretion initiated by vagal stimulation is not only high in acid but also high in pepsin (3, 13).

Clinically, it has been convenient to use the acidity of the gastric contents in quantity and concentration as a measure of the proteolytic activity of the gastric secretion, and most efforts at the treatment of peptic ulcer have been aimed at reduction or neutralization of the acidity of the gastric juice. It is generally forgotten that it is the pepsin activated by the acid which is the true measure of proteolytic activity and that the concentration and amount of acid present in the gastric secretion is not necessarily a true parallel of the proteolytic activity of that secretion (17, 18).

Most methods of treatment have been aimed at modifying or reducing gastric secretion without reference to the cause of the excess secretion in that particular case. Among the medical treatments diet and antacids have been used to neutralize excess acidity. Secretory depressants have proved useful. Rest (both emotional and physical) is widely recognized as beneficial—probably by reducing emotional stimulation of the vagus. Gastrojejunostomy, subtotal gastrectomy, and vagotomy have been most popular surgical procedures. Gastrojejunostomy is effective in many cases by relieving pyloric obstruction, and what is probably more important, by allowing a reflux of alkaline jejunal contents to neutralize gastric acidity. Subtotal gastric resection has aimed at suppressing the humoral secretion, and in addition, unless a Billroth I type of anastomosis is performed, takes advantage of the neutralizing action of gastro-

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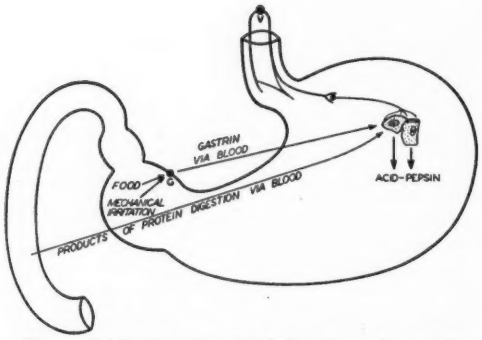

Fig. 1. Mechanisms for stimulation of gastric secretion. *V*, Origin of vagal impulses from the central nervous system. *G*, Site of formation of gastrin. This is limited to the pyloric part of the stomach. Nervous (vagal) phase of gastric secretion stimulated by: (1) emotional factors, (2) hypoglycemia, (3) taste, thought, or smell of food, (4) intracranial lesions. Humoral phase of gastric secretion stimulated by: (1) food in pyloric part of stomach, (2) mechanical irritation of pyloric part of stomach, (3) products of protein digestion after absorption by intestinal mucosa, (4) histamine, caffeine, or alcohol in blood or stomach.

jejunostomy (2, 39). It also removes a variable amount of gastric mucosa. In the last few years vagotomy has been popularized to abolish the vagal secretion.

The work of Dragstedt and others on sections of the vagus nerve has awakened a renewed interest in the physiological background of peptic ulcer and has stimulated a search for better methods of surgical treatment (25, 29, 35). There have now been enough vagus sections reported to indicate that neither vagotomy nor gastric resection alone is the final answer to all cases of peptic ulcer (1, 10, 38). From the recent literature it would appear that the recurrence rate of ulcer following either gastric resection or vagotomy is about equal. The undesirable side-effects of the two operations are comparable. The mortality rate following vagotomy is probably considerably less than that following gastric resection. Gastrojejunostomy is still a useful operation, either alone or in conjunction with subtotal gastric resection or vagotomy, although when used alone it does not give as high a percentage of cures as do either resection or vagotomy and is frequently followed by gastrojejunal ulcer.

With the foregoing facts in mind and the probability that there are two factors (vagal and humoral) concerned with the maintenance of peptic ulcer, it occurred to us that it would be useful to determine which secretory mechanism is predominant in a given case before carrying out any operative procedure in an attempt to abolish the abnormal mechanism. Actually the question is, should the humoral phase of gastric secretion be modified by subtotal gastric resection, or should the cephalic phase be abolished by vagotomy?

Our present concept of ulcer maintenance may be illustrated as in Figure 2. The line, ulcer threshold, is a level of mucosal resistance, including such factors as the amount of mucous and other protective mechanisms. This level probably varies in different patients and from time to time in the same patient. Gastric secretion, in amount and concentration exceeding this threshold, would cause maintenance of an ulcer. Spontaneous or seasonal remissions and exacerbations of peptic ulcer show that the ratio of proteolytic activity to mucosal resistance may vary from time to time. From the fact that the majority of ulcers may be healed by abolishing either the humoral or vagal phase (subtotal resection or vagotomy) it would appear that in most of them the situation would be somewhat as shown in column 1 where the suppression of either the humoral or vagal secretion would bring proteolytic activity below the threshold line. However, in column 2 in which the vagal secretion exceeds the threshold, it would appear that vagotomy would heal the ulcer while gastric resection would result in recurrence or gastrojejunal ulcer. Likewise, in column 3 vagotomy would be ineffective in controlling the ulcer while gastric resection would bring the column below the threshold line.

Working according to this hypothesis we have attempted to differentiate between the humoral and vagal factors in ulcer patients by performing preoperative studies of the comparative response to humoral and to vagal stimulation. The functions[1] tested are secretory rate, emptying rate (a function of gastric motility), peptic power, and least important

[1]The gastric function studies reported in this work were developed and carried out by Dr. Arthur M. Schoen of the Departments of Medicine and Pharmacology of the University of Louisville School of Medicine under a grant from the National Institute of Health.

of all, acidity. The secretory rates and emptying rates are calculated according to the methods of Schoen (31, 32). Peptic power is measured by a modification of the method of Mett (12), and the hydrogen ion concentration is measured electrometrically.

We have been able to differentiate, in the few cases thus far studied, between one group which seems to respond predominately to vagal stimulation and another group which seems to respond predominately to humoral stimulation as measured by secretory rate, emptying rate, and output of pepsin and hydrochloric acid. A third, and probably the largest group, seems to respond almost equally well to either stimulus. The following case reports represent an example from each group.

CASE 1. Predominate response to vagal stimulation (Fig. 3 and Table I). Patient L. S., 63 year old white male with symptoms of peptic ulcer disease for 30 years, had melena in November, 1947. He had 3 episodes of marked gastric retention between December, 1947, and April, 1948. There was fluoroscopic and x-ray evidence of crater in duodenal bulb, marked duodenal deformity, and 40 per cent retention of barium at 5 hours on November 24, 1947. Celiotomy, performed on April 29, 1948, revealed marked duodenal deformity with induration and scarring. Symptoms of gastric retention and pain were subsiding at the time of the gastric function studies on April 22, 1948. The average values of basal function during the three control periods were: peptic power, 4,000 units per cubic centimeter; free acidity, 34 clinical degrees (milliequivalents per liter); secretory rate, 0.77 cubic centimeter per minute; emptying rate, 1.21 cubic centimeters per minute. The average rate of secretion of free hydrochloric acid (hydrogen ions) was 0.0262 milliequivalent per minute, and the average rate of secretion of pepsin was 3,100 units per minute (not shown in Fig. 3).

The high peptic power of the control (basal) secretion is strongly suggestive of a high concentration of vagally stimulated gastric juice. The accompanying low rate of basal secretion of essentially normal acidity indicates a low output of hydrochloric acid and mucus during the control periods. The differences in emptying rates between the three control periods are within the range of a normal healthy individual.

Histamine had the usual influence on the rate of secretion of hydrochloric acid. The output of free hydrochloric acid was increased

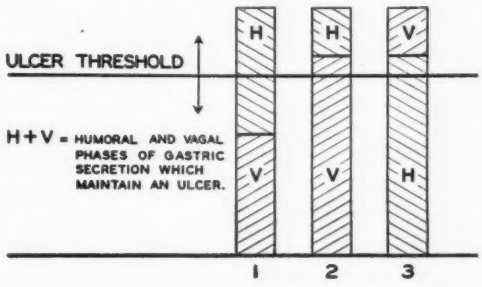

Fig. 2. Concept of peptic ulcer maintenance. When column exceeds the threshold, the ulcer is maintained. Patients represented by column 1 should benefit from either vagotomy or subtotal resection. Those represented by column 2 should benefit from vagotomy but not from subtotal resection. Those of column 3 should benefit from subtotal resection but not from vagotomy.

from 0.0262 to 0.563 milliequivalent per minute (not shown in Fig. 3). The acidity of the gastric juice was increased from 34 to 125 clinical degrees (milliequivalents per liter). While the peptic power of the gastric juice was diminished following the injection of histamine, the actual output of pepsin from the mucosa was increased during the first period after the histamine injection (18¾ minutes) from 3,100 to 8,000 units per minute (not shown in Fig. 3). This increased output of pepsin by the gastric mucosa following the injection of histamine has been attributed to the "washing-out," by the increased secretion of the parietal cells, of the gastric crypts and alveoli which contained a normal accumulation of secretion from the acid and pepsin secreting cells. The rate of pepsin output at the time the maximum rate of histamine stimulated gastric secretion occurred was only 1,580 units per minute. The increase in the rate of gastric emptying was due partly to the increased volume of gastric contents as a result of the increased secretory rate. There was an initial depression of the emptying rate following the injection of histamine.

Insulin hypoglycemia produced a much higher rate of gastric secretion and emptying than did histamine. The increase of gastric function was preceded by a depression of all components. This latter phenomenon has occurred consistently in all people after the injection of insulin. The maximum rate of pepsin output during insulin hypoglycemia was

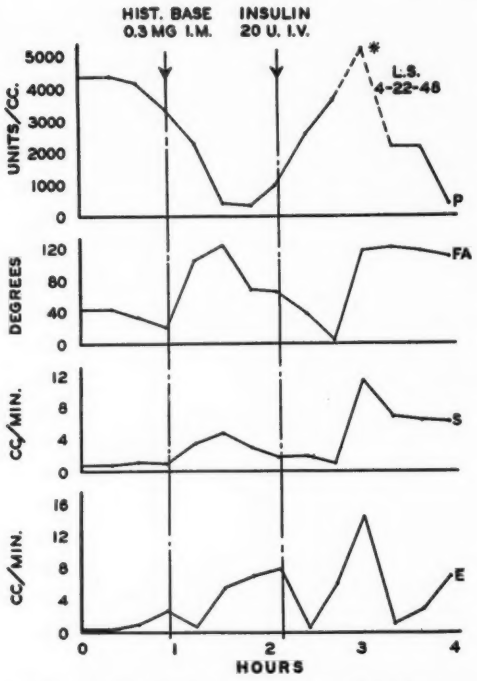

Fig. 3. Gastric function of one whose predominate response was to vagal stimulation. *P*, Peptic power of residual gastric juice at end of test period adjusted to *p*H 1.68. *FA*, Free acidity of residual gastric juice at end of test period. *S*, Average secretory rate during test period. *E*, Average emptying rate during test period. *, Albumin tubes were digested completely; minimum peptic power, 5,200 units per cubic centimeter.

60,400 units per minute which is far in excess of either the basal rate (3,100 units per minute) or the maximum rate following the injection of histamine (8,000 units). This maximum rate of pepsin output occurred during the period when secretory rate was also a maximum.

Although the maximum concentration of free hydrochloric acid in the histamine stimulated gastric juice was essentially the same as the maximum attained during hypoglycemia (125 and 124 clinical degrees), the maximum rate of secretion of free acid following histamine was 0.563 milliequivalent per minute as compared with 1.380 milliequivalents per minute during insulin hypoglycemia (not shown in Fig. 3).

TABLE I.—SUMMARY OF THE RESPONSES OF GASTRIC FUNCTION TO HISTAMINE AND INSULIN HYPOGLYCEMIA

Case No. Initials	Average values of basal function during 3 control periods	Average values for period of maximum response to histamine	Average values for period of maximum response to insulin
Peptic power[1], units per c.c.			
1 L.S.	4000	2300	3700
2 E.W.	0	780	0
3 A.M.	1600	2100	2600
Pepsin output[2], units per min.			
1 L.S.	3100	8000	60400
2 E.W.	0	2540	0
3 A.M.	1660	4500	7050
Pepsin output when secretory rate was maximum, units per min.			
1 L.S.	4000	1580	60400
2 E.W.	0	2540	460
3 A.M.	1250	3200	7050
Free acidity[3], clinical degrees			
1 L.S.	34	125	124
2 E.W.	0	54	0
3 A.M.	29	85	86
Free acid output[4], mEq. per min.			
1 L.S.	0.0262	0.563	1.380
2 E.W.	0.0	0.175	0
3 A.M.	0.0302	0.340	0.224
Secretory rate, c.c. per min.			
1 L.S.	0.77	4.50	11.6
2 E.W.	1.15	3.25	1.0
3 A.M.	1.04	4.00	3.1
Emptying rate, c.c. per min.			
1 L.S.	1.21	7.78	14.6
2 E.W.	1.90	2.40	1.0
3 A.M.	2.24	4.00	4.9

[1] 1 unit per c.c.—same as Mett unit except that exponent of 2.5 was used for calculating peptic power.
[2] Pepsin output—peptic power × secretory rate.
[3] 1 clinical degree—1 milliequivalent of acid per liter of gastric juice.
[4] Free acid output—free acidity × secretory rate.

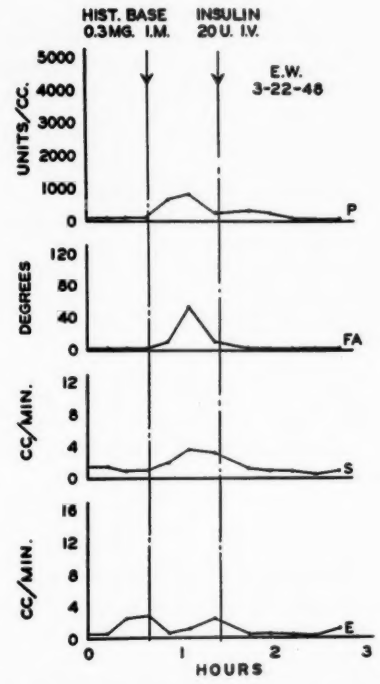

Fig. 4. Gastric function of one whose predominate response was to humoral stimulation.

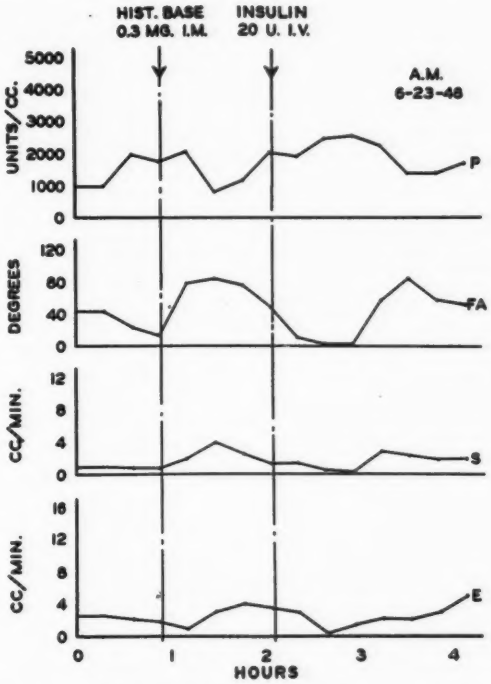

Fig. 5. Gastric function of one whose response was about equal to vagal and humoral stimulation.

CASE 2. Predominate response to humoral stimulation (Fig. 4 and Table I). Patient E. W., 62 year old white male with recurrent symptoms of peptic ulcer for more than 6 years, showed evidence of complete obstruction in February, 1948. X-ray examination on March 15, 1948 showed 90 per cent retention of barium at the end of 5 hours. Celiotomy, performed on March 26, 1948, revealed evidence of duodenal ulcer and marked scarring of the first part of the duodenum. The pyloric obstruction was subsiding at the time of the gastric function studies on March 22, 1948. The average values of basal gastric function during the three control periods were: peptic power, 0 units per cubic centimeter; free acidity, 0 clinical degrees (milliequivalents per liter); secretory rate, 1.15 cubic centimeters per minute; emptying rate, 1.90 cubic centimeters per minute.

The absence of active pepsin and hydrochloric acid (*p*H of the basal secretion was 7.0) in the gastric juice of the control periods suggests that the nervous phase of gastric secretion was acting not at all or very little. With no food in the stomach or intestines, the humoral phase also would not be acting, hence the absence of acid and pepsin in the basal secretion. A small amount of acid or pepsin could have been neutralized by mucous secretion.

Histamine increased the secretory rate and stimulated the production of hydrochloric acid. The response to histamine in this patient was less in every respect than the response in patient L.S. (Fig. 3). The presence of pepsin in the histamine stimulated gastric juice, again could be due to the "washing-out" process mentioned under Case 1. This would imply, however, that some pepsin had been present in the gastric alveoli previous to the injection of histamine. Whether it had been produced during the basal periods by either the nervous or humoral phases cannot be determined. Histamine again caused an initial depression of the emptying rate which was followed immediately by an increased rate of emptying.

During the 10 minute period immediately following the injection of insulin, there was a marked decrease in all components of gastric function. This phenomenon occurs whenever insulin is injected. The increase of gastric function which usually follows the initial period of inhibition did not occur in this person (Fig. 3). Instead, there was a persistence of the state of diminished gastric activity. The initial increase of the peptic power following the injection of insulin resulted from the decreased secretory rate and does not represent an increase in the output of pepsin. Actually, the output of pepsin dropped from 570 units per minute to 460 units per minute during the 10 minute period following the injection of insulin, and it continued to drop to zero (not shown in Fig. 4).

CASE 3. Equal response to vagal and humoral stimulation (Fig. 5 and Table I). Patient A. M., 47 year old white male with history of peptic ulcer for 10 years had a massive hemorrhage in April, 1948. X-ray examination and fluoroscopy showed crater in duodenum, and clover-leaf deformity of the pyloric cap on June 22, 1948. Typical symptoms of peptic ulcer were subsiding at the time of the gastric function studies on June 23, 1948. The average values of basal gastric function during the three control periods were: peptic power, 1,600 units per cubic centimeter; free acidity, 29 clinical degrees (milliequivalents per liter); secretory rate, 1.04 cubic centimeters per minute; emptying rate, 2.24 cubic centimeters per minute. The average rate of secretion of free hydrochloric acid (hydrogen ions) was 0.0302 milliequivalent per minute, and the average rate of secretion of pepsin was 1,660 units per minute (not shown in Fig. 5).

The lower concentration of pepsin in the basal secretion of this person as compared with the pepsin concentration in the basal secretion of patient L.S. (Fig. 3) suggests a lower concentration of vagally stimulated gastric juice. The pepsin output of this patient under basal conditions was 1,660 units per minute which is less than one-half of the pepsin output of patient L.S. under similar conditions. A comparison of the acidities and secretory rates of the basal secretions of this person with patient L.S. suggests that in this person there was relatively a higher rate of secretion of nonacid and nonpepsin constituents (mucous secretion).

Comparisons of the maximum responses to histamine and insulin show that histamine stimulated the secretion of hydrochloric acid slightly more than did insulin hypoglycemia (0.340 milliequivalent per minute for histamine, and 0.224 milliequivalent per minute for insulin hypoglycemia). Secretory rate also was stimulated somewhat more by histamine (4.0 c.c. per minute for histamine, and 3.10 c.c. per minute for insulin). However, peptic power, output of pepsin, and emptying rate were all stimulated more by insulin (Table I).

We are now basing our operative therapy on the differentiation of duodenal ulcer patients into the groups as shown by these tests, doing subtotal resections on those patients who respond predominately to humoral stimulation and vagotomy on those responding predominately to vagal stimulation. In the third group, in which it would seem that equally good results would follow either procedure, the choice of operation may depend upon factors other than the gastric function studies. The gastric resections performed are the standard high resections with care to remove all antral tissue. Continuity is usually re-established by anterior Polya gastrojejunostomy. Vagotomy is carried out transthoracically unless there is a special indication for abdominal exploration or unless gastrojejunostomy is to be added to the vagotomy. No gastric ulcers are included in this study since we believe that the incidence of unsuspected carcinoma in gastric ulcer is so high that any procedure which does not involve resection of the ulcer is dangerous. It is also believed that gastric ulcer results from a different condition than duodenal or jejunal ulcer, not having the same age incidence, sex incidence, or necessarily the same physiological characteristics. Gastrojejunostomy is performed in addition to vagotomy on all patients whose stomachs are not empty at the end of 2 hours following the ingestion of barium. While it is realized that a normal stomach may retain barium as long as 5 hours, it is felt that in the hypertonic, hypermotile stomach with ulcer any retention beyond 2 hours indicates relative pyloric obstruction and that symptoms of retention may occur unless a drainage operation is combined with the vagotomy. It has been argued that the addition of gastrojejunostomy prevents a thorough evaluation of vagotomy. On

the other hand, gastric resection of the Polya, Hofmeister, or similar types, also takes advantage of gastrojejunostomy by bringing the alkaline jejunal contents to the most vulnerable spot, which is the area of the anastomosis.

It is evident that it will require many cases handled according to this method followed over a period of years to test the validity of our hypothesis. This work is presented in its present early stage in the hope that others who are interested in the problem of peptic ulcer may work along similar lines to prove or disprove this hypothesis in a shorter time than we would be able to do alone and to develop practical tests of gastric function which will give more adequate information in planning a rationale of treatment than have the tests which are in general use.

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THE MECHANISM OF THE INTERVERTEBRAL DISC PROTRUSION

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THE concept of sciatica has changed so radically and rapidly in the 15 years since Mixter and Barr's basic contribution to its diagnosis and surgical treatment that pathological anatomy and physiology have been unable to keep up with the clinical development.

Both sciatica and low back pain—the scourge of neurologists and orthopedic surgeons for scores of years—seem to have become a problem for the neurosurgeon. However, growing experience has shown that operative treatment does not benefit, in the long run, more than one-half to two-thirds of the patients in this group. Indeed, the fact that the neurosurgeons have changed their operative approach and technique repeatedly would suggest that they were not wholly satisfied with their results. It is quite confusing to see not only that the success of the same operative method varies widely in the hands of different surgeons but also that a method is sometimes dismissed by the same surgeon who had praised it previously, because he found it ineffective in some patients.

It has been suggested that valid statistics of the final results of an operative method will be obtained only if it is performed by the same surgeon with controls for comparison in a sufficient number of patients taken at random. However, this method of sampling will give the expected results only if the basic pathology is uniform, and that is exactly what is under scrutiny. If the presumption is made that the mechanism of disc protrusion is not the same in all patients, then it is obvious that one type of treatment will not fit all patients.

Most conjectures concerning the mechanism of the intervertebral disc protrusion are not based on objective experience with the pathological process. A great amount of detailed knowledge has been collected as to the clinical, x-ray, and operative findings in the disc syndrome but very little is known about the pathological anatomy of the process. A few operative and many diagrammatic sketches have been published indicating what was seen at operation and what the surgeon inferred as to the mechanism of the intervertebral disc disorder. Very few of these concepts were based on histologic-pathological findings. Not even a uniform nomenclature has been generally accepted. The terms hidden or bulging disc, ruptured, herniating, protruded, and slipped disc are sometimes used as if interchangeable. In reality, they denote entirely different conditions.

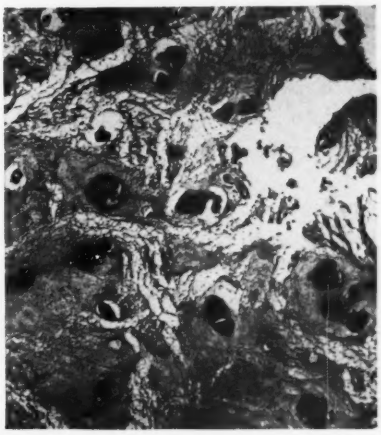
Statistics concerning 40 cases of disc removal for sciatica from patients whose clinical, x-ray, operative, and pathological data were complete and who could be followed-up for a sufficient number of years form the basis of Table I. These cases represent part of a group of 169 in which removal was carried out by F. C. Grant and R. A. Groff in the Hospital of the University of Pennsylvania and the Graduate Hospital in Philadelphia respectively, between the years 1939 and 1943.

TABLE I.—TYPES OF DISCS OPERATIVELY REMOVED

Types	No.	Per cent	Good or better results		Average result of the whole group
			No.	Per cent	
Bulging	15	37.5	5	33.3	Fair to poor
Herniating	11	27.5	9	82	Excellent to good
Slipped	14	35	8	57	Good to fair
Total	40				

Histopathological examination of the 169 discs removed at operation and of 20 controls seems to suggest that the pathological findings may be grouped into three almost equal classes with widely different results.

From the Neuropathological Laboratory of the Neurosurgical Service of the University Hospital and the Department of Neuroanatomy of the Graduate School of Medicine, University of Pennsylvania.



Figs. 1 and 2. S. B., 42–132. Elastica, Fig. 1, left, × 16, Fig. 2, × 88. Origin: traumatic. Operation: revealed a ruptured disc at the fourth lumbar vertebra. Pathologically, the lamellae of the annulus fibrosus are partly preserved, partly destroyed. The inner lamellae are increasingly necrotic (*IL*, Fig. 1). The disc proper shows dense hyaline cartilage with gradual thinning into fibrous cartilage and reticular dissolution around numerous large cartilage cells (Fig. 2). No part of the nucleus pulposus was found. Result: good in the beginning; 5 years after operation, patient complained of moderate sciatic pain in the left buttocks and was dissatisfied with the result.

GROUP I

The poorest results were found in the 15 patients of our first group, namely in discs which the surgeon described as *bulging*. This term is meant to indicate that the posterior part of the annulus fibrosus protruded upward without any visible lesion of the annulus or the cartilage. This form seems to correspond with what has been called "hidden disc." It was assumed that this anomaly was the cause of the clinical symptoms and signs. Hence, the annulus was incised and the allegedly diseased disc material removed.

Histopathological examination of the biopsy material fails to show any abnormality. It contains normal lamellae of fibrous cartilage sometimes with adherent gelatinous tissue.

Results: Not more than one-third of the persons in this group experienced reasonable benefits from operation. The inference is that in those not improved the bulging annulus had not been the cause of the complaints. Gurdjian[1] states that in his experience persons with bulging disc and good operative results had shown invariably the typical defect in the myelogram and had suffered traumatic lesions of the disc lamellae without a visible rent in the annulus. Mixter[1] takes exception to the last part of this opinion. He believes that a small rent of the annulus may be easily overlooked unless the patient's spine is overstretched during operation.

A bulging disc results from a paunchy posterior annulus fibrosus without rent in the annulus and without detachment of the disc from the bone. It should not be incised unless there is definite evidence of its encroachment upon a posterior root.

GROUP II

In our second group, a rent in the annulus fibrosus was found during operation through which disc material was *herniating* into the spinal canal. A spinal root was found hooked up over it.

Histological examination of the biopsy material reveals cartilaginous lamellae of the disc in varying degrees of necrosis and liquefaction (Fig. 1). The inner lamellae of the disc are often necrotic. Dense hyaline cartilage is seen gradually thinning out into fibrous cartilage with reticular dissolution of the tissue around large cartilage cells (Fig. 2). Oc-

[1]Personal communications.

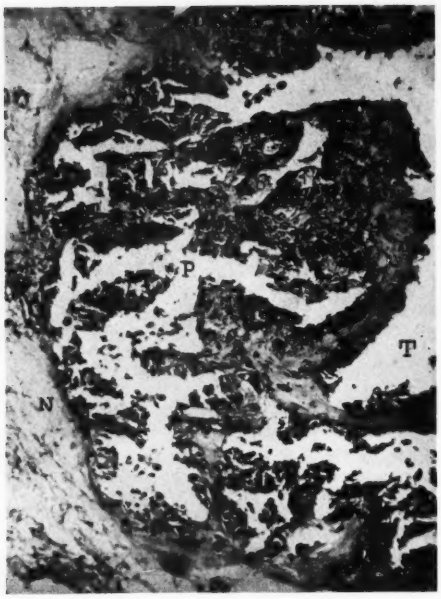


Fig. 3. H., 42–112. Elastica, × 88. Origin: traumatic. Clinically: typical sciatica. X-ray: spondylolisthesis. Operation: revealed a small disc consisting of a small rim of reticular hyaline cartilage surrounding the nucleus pulposus. The nerve was found abnormal and freed. Pathologically an advanced granular-reticular necrosis (*N*) of the feathery gelatinous substance surrounding the pulp was found. An unusually large portion of the nucleus pulposus was adherent to the disc. The picture shows the typical tuft-like structures (*T*) lined by cartilage cells, some of them in gelatinous degeneration. Result: good.

casionally, an amorphous mass is found which may represent liquefied cartilage or may constitute part of the nucleus pulposus (Fig. 3). However, there is no evidence that the hernia usually consists of nucleus pulposus. In other instances, the rent does not go straight inward but obliquely upward, severing the attachment of one or more lamellae to the epiphyseal plate (Fig. 4). Pieces of brown atrophy lined by connective tissue are sometimes visible and may indicate the long duration of the process (Fig. 5).

Result: Removal of the herniated tissue and freeing of the involved posterior root gave excellent or good results in 82 per cent of those operated upon in this group.

A herniating disc results from traumatic necrosis of the disc lamellae and their protrusion through a rent in the annulus fibrosus. Liquefied cartilage or viscous material of the nucleus pulposus may be extruded. The protruded mass has to be excised and the content of the disc eventrated sufficiently to free the overextended posterior root. The good operative results indicate that the surgical procedure was adequate in this group.

GROUP III

In our third group of patients, it was found that an eccentric posterior portion of the disc had actually *slipped* backward from its attachments to the bony surface of the vertebral bodies. In other words, this type represents a true focal protrusion of the intervertebral disc. It is not easy to explain satisfactorily from the operative appearance of the disc or from gross examination of the specimens the mechanism of "slipping." The apparent reason for this is that the surgeon has to cut into the protruding material in order to remove it. The direction of the incision is dictated by the narrow space of exposure rather than the anatomical situation. Only when the slipped portion of the disc was broken off and removed in its entirety, was a revealing specimen made available.

Pathological examination shows a picture entirely different from that of the previous group. We find parts of the epiphyseal plate and sometimes of the bone itself (Fig. 6). A group of loosely arranged bone septa is present, sometimes filled with red bone marrow, fragments of the posterior longitudinal ligament and cartilaginous lamellae of the disc. The continuity of bone, epiphyseal plate, hyaline, and fibruous cartilage is evident. The tissue may be in degeneration as is to be expected after being separated for quite a while from its normal environment. The fact that this often gives the inner part of the disc a gelatinous appearance but that no tufts or cartilage cells are visible suggests that the tear has been confined to the posterior one-fourth or one-third of the disc.

A similar though less complete pathological picture is found if the protruded disc has not been fractured and the annulus has had to be incised to remove the injured lamellae. Again we find a large portion of the disc with its at-

Fig. 4.

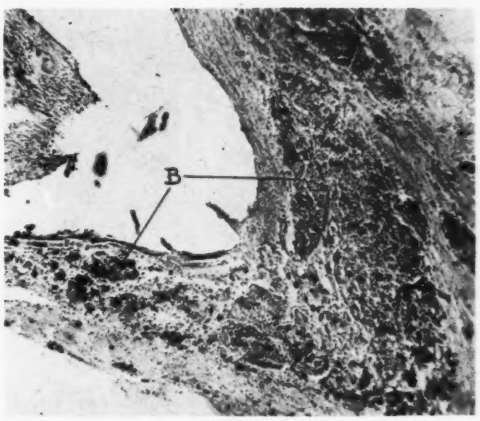


Fig. 5.

Figs. 4 and 5. A., 50 years, 42–146. Fig. 4, phosphotungsic acid hematoxylin, × 88; Fig. 5, van Gieson, × 8. Origin: no trauma remembered. Clinically: typical sciatica of 20 years' duration. Operation: revealed a large disc over which the nerve was hooked up and freed. Pathologically the piece removed represents a section of the intervertebral disc (Fig. 4). The surface of its bony attachment is only .6 centimeter long but its extension toward the center of the disc is more than 1 centimeter long. It shows clearly from above downward the ragged bony attachment of hyaline cartilage (*H*) followed by fibrous material but no pulp. The detail (Fig. 5) shows patches of brown atrophy (*B*) and a few strands of connective tissue fibers at its inner margin. Result: good.

tachment. The line of cleavage, as a rule, runs smoothly through the hyaline cartilage (Fig. 7), or particles of bone may be found in a ragged line of separation (Fig. 8). In either instance, the disc is characterized by its horseshoe shape which results from the elasticity of its stiff fibers after their separation from the bone. The cartilaginous lamellae are cross-sectioned so that one layer after the other is visible, from the rest of the annulus fibrosus on the left to the more central portions of the disc on the right (Fig. 7). Areas of partial necrosis and softening and others of granular degeneration are visible in the fibrous part of the disc. Only occasionally remnants of cartilage cells or other parts of the nucleus pulposus are found. In other words, the histological sections show a radial slice of a disc transsecting the lamellae in a vertical sector and running parallel with the cartilage fibers.

Results: The fact that only 57 per cent of those operated upon in this group had a good result suggests that the surgical technique may be improved.

In slightly more than one-third of our cases of disc removal, injury led to rupture of the lamellae extending into the epiphyseal plate and to fracture of the rim of the vertebra. This lesion frees the disc from its anchorage and permits it to slip backward. In doing so it encroaches upon a root and extends it. The sharp bony margins may secondarily sever the slipped portion of the disc partially or completely.

DISCUSSION

Various questions pertaining to the disc syndrome have been raised. The most puzzling of all is what happened to the victims of sciatica who were not operated upon prior to the last 15 years? True, the neurologist saw patients with chronic sciatica. But with our present experience, one would expect large numbers of them eventually to enter institutions for the chronically sick. This was definitely not the

Fig. 6. Sister R., 42–14. Elastica, × 8. Origin: traumatic. Clinically, typical sciatica. X-ray: upper posterior angle of the fifth lumbar vertebra broken off. Operation: revealed a ruptured disc. The root was hooked up and freed. Pathologically, the picture shows the posterior lower angle of the fifth lumbar vertebra with broken down bone lamellae (*B*). The posterior longitudinal ligament (*PL*) is adherent to the bone at its posterior (right) margin, the hyaline and fibrocartilaginous tissue of the intervertebral disc (*D*) to its inferior surface. Only a few ring-shaped lamellae of the disc are preserved as dark lines. Most of the tissue shows a hyaline degeneration which becomes slightly gelatinous toward the center of the disc. Result: excellent for 2 years until she began strenuous physical work resulting in instability of her back. She was working without complaints while wearing her brace but fusion was recommended by orthopedists. Went to India for missionary work without fusion. Satisfied 7 years later.

case. There is good reason to believe that parts of the protruded disc become separated and resorbed in the course of time. One also wonders how it happens that in a considerable number of husky persons trivial trauma leads to infraction not only of the epiphyseal cartilage but also of the bone. If, however, this mechanism is operative, why is it found in only about one-third of the patients with disc syndrome?

The majority of both neurologists and neurosurgeons agree today that *not* all cases of sciatica are caused by a protruding disc or lend themselves equally well to operative treatment. Consideration of the anatomical and pathological findings indicates clearly that sciatic pain—in contrast to the pure low back pain—cannot be blamed on an abnormal disc unless a posterior root is compressed by it. Decompression of the involved root is, therefore, the first requisite of a successful operation for sciatica. This explains clearly why a simple *bulging* disc should be left alone.[1] The validity of this statement may be strengthened by the fact that bulging discs were found in necropsy in 28 per cent of 75 persons who at no time had complained of sciatica (Horwitz).

In considering the postoperative results, the patients of this, more than the other two groups, complained of backache rather than true sciatica. Our material is too small for a conclusive estimate but if our impressions were correct, the widsom of removing parts of a normal disc would be further challenged, because an already unsatisfactory stability of the spine would be disturbed to a still greater extent. A more rational procedure in this group of patients might be fusion of the spine, so highly recommended by orthopedic surgeons, yet not found to be superior to simple disc removal by neurosurgeons.

There can be no doubt that disc material *herniating* through a rent in the annulus fibrosus needs thorough removal to free the involved posterior root. But again the question arises: What is the rationale, from the pathological point of view, of scraping out from within the disc all available material or of attempting evacuation of the nucleus pulposus? Finally, why does the annulus fibrosus, surrounding the disc, rupture just at its posterior circumference?

These questions cannot be answered without a true three dimensional concept of the configuration of the normal intervertebral disc together with its attachment to its adjacent vertebrae.

It has been said that the intervertebral disc functions as a water cushion between two vertebrae. This is obviously no more than an

[1]Spurling and Grantham have recently stressed these two points.

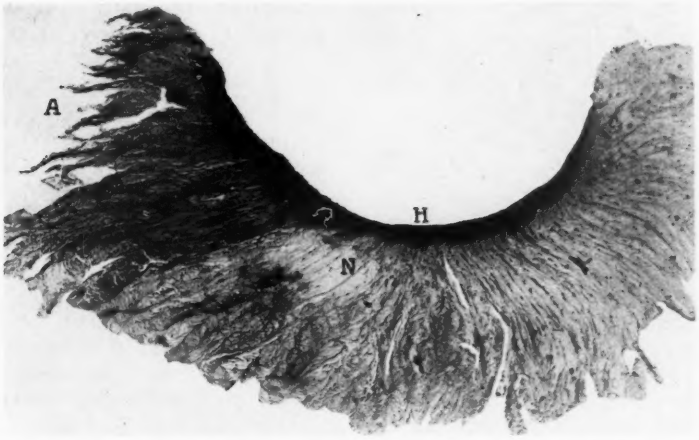


Fig. 7. H. B., 42–91. Elastica, × 9. Origin: traumatic. Clinically: low back pain, no sciatica. X-ray: epiphyseal rupture. Operation: revealed a protruding disc which was removed after incision of the annulus fibrosus. The hooked-up nerve was freed. Pathologically, the disc showed bony adhesions at the hyaline (*H*) cartilaginous layer, a large fibrous layer with some brown and granular degenerations and necrosis in the transitional area to the central part (*N*). *A*, Annulus fibrosus; no pulp. Result: excellent.

Fig. 8. Mrs. B. Th., 60 years, 47–179. van Gieson, ×9. Origin: unknown. Clinically: incomplete sciatica. X-ray: narrowing of interspace between the fourth and fifth lumbar vertebrae. Slight spondylolisthesis of the fifth lumbar. Myelogram shows posterior displacement of disc. Operation: revealed a horseshoe shaped disc below the fifth lumbar. The dura was incised and a considerable amount of disc material was removed. The disc consisted pathologically of hyaline cartilage with attached bone fragments and fibrous cartilage containing up to 8 large boxed-in cells. No pulp was adherent. Result: 2 years later, numbness and paresthesia were still present in the left foot. Pain was shooting down the left leg. Lasègue positive. Patient believed to be improved by 80 per cent but this is in contrast to the objective findings.

analogy but it molds our mental image of the anatomical structure of the disc suggesting that it is a bladder with more or less solid walls and a viscous core containing the resid-

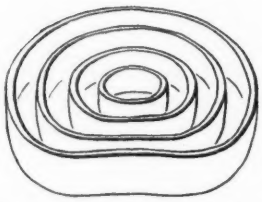


Fig. 9. Diagram of the structure of the disc lamellae.

uals of the notochord. Some technical operative recommendations seem to stem from this erroneous concept.

Actually, the disc is composed of numerous cylindroid-shaped lamellae arranged around one another in near concentric fashion (Fig. 9). The greater part of these lamellae consists of fibrous cartilage. The lamellae fuse together at their upper and lower ends in a homogeneous plate of hyaline cartilage (Fig. 12). These plates represent simultaneously the upper and lower surfaces of the disc and the epiphyseal plates by means of which the disc is attached to the adjacent vertebrae. The external layers of the lamellae which form the circular wrappings of the disc—the annulus fibrosus—are strongly fibrous and stiff. They do not fuse into the epiphyseal plate but are directly inserted into the bone surface of the vertebrae (Sharpey's fibers).

Fig. 10. L., 42 years, colored, 42–135. Elastica, ×88. Origin: traumatic. Clinically: typical sciatica. X-ray: sclerosed aorta. Operation: herniating disc. Pathologically, severe reticular atrophy of fibrous cartilage with large fibrosed cartilage cells. This is a progressed state of degeneration, possibly of vasculonutritional origin, characterized by absence of viable cells within the boxes. Some boxes are filled with connective tissue, others are empty. Result: seemed good in the beginning but was unsatisfactory 5 years later, probably because of the insufficient blood supply from the sclerosed aorta.

The central portion of the disc consists of a biconvex, lentil-shaped mass—the nucleus pulposus—which is gelatinous in youth and becomes more fibrous in adult life. Peterson describes the nucleus pulposus as "true fibrocartilage in an almost fluid interfibrillary substance," and Schaffer as "chrondro-mucoid jelly." Soft fibrous cartilage lining the nucleus pulposus protrudes tuft or feather like into its ragged cavity. Notochord cells are found in its center at birth but disappear after the third year of life. Fick, Saunders, Dockerty and Love have described and illustrated vesicular cells in the pulp of adults which they identify with notochord cells. However, most anatomists and pathologists agree that no notochord cells survive in adults unless in a true chordoma. The cells seen in the gelatinous part of the disc are surrounded by a halo and arranged in groups of 2 or 4 within a "mother capsule" (Koelliker). They represent cartilage cells.

Small foci of softening and cavity formation in the fibrous cartilage seem to occur normally. Conversely, wear and tear in consequence of strain and age seem to change the disc in the opposite direction. The nucleus pulposus loses its viscous appearance and becomes more fibrous, tendinous, chondroid, and eventually calcified (Uebermuth). Rathcke found gross calcification of the nucleus pulposus in 4.1 per cent of spines in patients between the ages of 30 and 50, and 8.7 per cent in the older age group. A powder-like calcium deposit is often seen on the tufts of the nucleus pulposus of older persons. The disc lamellae shrink and seem to be more brittle. Signs of brown atrophy are found. However, no indication of disease was observed in our disc biopsies with the three following exceptions. Marked necrosis was present in the discs of 3 middle aged colored men with x-ray evidence of progressive sclerosis of the descending aorta (Fig. 10). They were considered as of vasculonutritional origin. Infection, inflammation, and cartilaginous metaplasia as described by Saunders and Inman were absent in our material. Schmorl believes that they are very rare in adults.

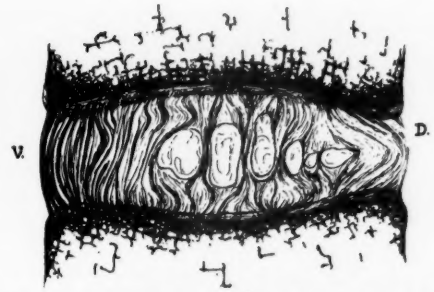


Fig. 11. Cross section of normal disc with separate nuclei pulposi, extending backward to dorsal aspect (*D*), and adjacent vertebrae. Modified after Boemig.

All authors agree that necroses with liquefaction of cartilage are common in biopsy material (Smith, Saunders and Inman) and fairly frequent in necropsy material (Schmorl and Junghans). However, there is difference of opinion as to cause and effect. Some believe that the disc ruptures because its lamellae have become necrotic in consequence of disease, others see in the necrotic tissue the effect of trauma. Hence, the first group is in favor of thorough surgical dismemberment of the disc to prevent further expulsion of diseased lamellae. Some are of the opinion that the extruded liquefied tissue is necessarily part of the nucleus pulposus. They speak of protrusion of the nucleus pulposus and go so far as to attempt scooping out of the entire central part of the disc. In our own biopsy material of herniating discs, tufts, capsule cells, or other characteristics of the nucleus pulposus were usually absent in the removed amorphous viscous material.

The position of the nucleus pulposus within the normal disc plays a major role in the theories of the protruding disc. Luschka described as a normal variant of the nucleus pulposus a posterior recessus which would bring the gelatinous mass close to the annulus fibrosus. However, Schmorl saw Luschka's posterior recessus in only 15 per cent of 400 normal spines, although segmentation of the pulp and even 2 or 3 independent gelatinous masses occurred in 75 per cent of 95 normal discs (Fig. 11, Boemig). Horwitz found unilateral posterior dislocation of the nucleus pulposus in 5.25 per cent of 75 normal discs.

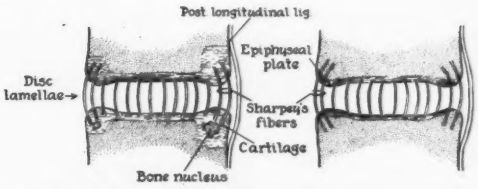


Fig. 12. Diagrammatic picture of anchorage of disc lamellae in epiphyseal plate. Bone: dotted; cartilage: horizontally dashed. The left picture is of a juvenile individual in whom the expanded rim of the vertebrae is still cartilaginous and contains the ring shaped bone nucleus. The Sharpey fibers of the annulus fibrosus are inserted deeply in the cartilaginous rim. The right picture shows the situation in the adult. The expanded rim of the vertebrae is ossified. Sharpey's fibers persist in the bone whereas the disc lamellae remain anchored to the small, permanently cartilaginous epiphyseal plates. Modified after Schmorl.

Schmorl whose opinion is based on the investigation of 8,000 spines summed up his experience in 1930: "As I become more familiar with the discs and their pathological changes, I am more and more convinced that their tissue is most delicate and vulnerable. It is frequently damaged by strain almost within the range of normal movements or by minor external force." Anatomical studies indicate furthermore that the annulus fibrosus is less dense posteriorly than anteriorly and laterally and finally, that the disc itself is less firmly fixed in the bone at the posterior circumference of the

Fig. 13. Sagittal section of normal intervertebral disc showing the adjacent vertebrae with the incomplete ossification of their rims. *a*, Longitudinal ligament; *b*, fibrous cartilage; *c*, hyaline cartilage; *d*, bone lamellae (after Petersen).

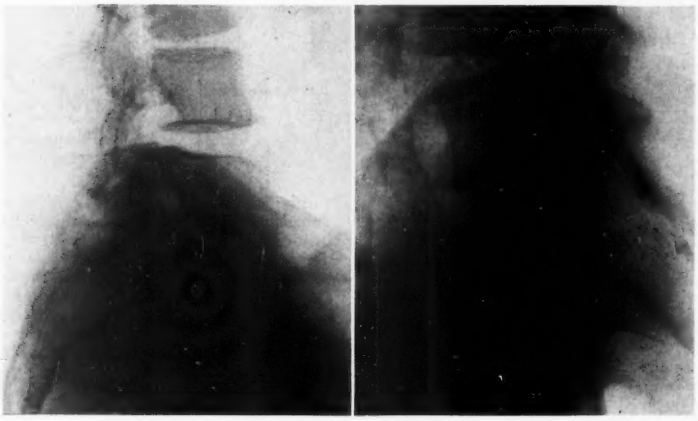


Fig. 14., left. H. K., 3045. Clinically: pain in the right back radiating down the right leg for 5 months. Poker back, Lasègue on right, bilateral absence of Achilles reflex: traumatic origin. X-ray: Narrowing of fifth lumbar interspace, lipping of bodies between the fourth and fifth lumbar vertebrae. Rupture of posterior lower epiphyseal rim of the fifth lumbar. Fifth lumbar, 17 to 15 millimeters. Spondylolisthesis of fifth lumbar. Myelogram: filling defect between the fourth and fifth lumbar vertebrae on the right. Operation: a loose cartilage was found and removed. Pathologically, this appeared to be a ruptured fibrocartilaginous disc consisting of part of the annulus fibrosus with adherent layers of straight and loose lamellae. Result, excellent.

Fig. 15. R., 1693, 58 years. Clinically: traumatic origin, incomplete sciatica. X-ray: narrowing of the fourth lumbar interspace. Clip outside. Collapse of upper posterior angle of the fifth lumbar with epiphyseal fracture. Wedging of the fifth lumbar to the degree that the posterior half of the fifth lumbar is only about three-fourths as thick as its anterior half. Fifth lumbar, 21 to 17 millimeters. Calcification of aorta. Operation: showed a herniating disc. The annulus fibrosus was incised and the disc curetted out. The nerves were freed. Histologically, this is a well preserved disc with complete layers of hyaline and fibrous cartilage, in the latter minor reticular rarefications. Result: good. Check-up 5 years after operation: No subjective complaints. Patient is satisfied.

vertebra. If we combine these considerations with the normal architecture of the disc lamellae it is easily understood that mild torque of the spine may lead to rupture of lamellae of the disc with subsequent necrosis and possible liquefaction. The debris may be pressed through a dehiscence of the annulus fibrosus. This mechanism explains why portions of the annulus are comparatively rarely found in the biopsy material and that the torn annulus often tends to fall back in place as soon as the nuclear part of the protrusion is removed (Bradford and Spurling). One may wonder under these conditions whether excision of more of the annulus and disc lamellae than is traumatized and necrotic anyway, will be of help to the patient. Certainly, not much can be expected in this instance from packing the cavity with bone chips since there is no free bone surface to which they may become attached. Finally, no good reason for fusion of the spine can be seen in the group of the herniating disc since the results of its simple removal are very satisfactory.

One third of our patients with disc syndrome had sustained a fracture of the epiphyseal plate with or without a chip fracture of the adjacent vertebral body. This is an extraordinary experience in a group of healthy individuals undergoing sometimes so mild an injury that it is difficult to remember it. The explanation may be seen in the normal development of the surface of the vertebral body. In the early years of life, the upper and lower expansions of the vertebrae are not ossified. A cap of cartilage is glued over them. Its inner surface represents the permanent epiphyseal plate while its sides cover the still re-

cessed rim in a circular fashion (Fig. 12). A ring shaped bone nucleus embedded within the cartilage builds eventually the bony expansions of the vertebral surfaces. However, this process may remain incomplete in otherwise normal persons. Large and small cavities are extant between flimsy bony septa (Fig. 13). Hence, this part of the vertebra is most endangered by brusque changes in position of the spine. It is true, the disc is protected by the longitudinal ligaments but the posterior ligament is not attached to the vertebra proper and only loosely to the annulus fibrosus. Thus, in addition to the universal greater vulnerability of the posterior aspect of the disc, an individual constitutional factor has to be considered, namely, the insufficient ossification of the vertebral rim.

Were the protrusion of the disc symmetrical, its attachments had to be broken off both at the superior and inferior epiphyseal plates all around the posterior circumference and at least in part at the lateral surfaces. I have not seen a histological specimen containing the two opposite epiphyseal plates and I have not heard that a surgeon has described the protrusion of a disc all around the posterior circumference. The histological picture speaks in favor of a vertical or oblique infraction of the epiphyseal plate and the posterior angle of the bony rim with loosening of a comparatively small sector rather than a horizontal fracture with expulsion of a large posterior semilunar section of the disc. Such an eccentric detachment and protrusion of the disc easily overtaxes the elasticity of the fibrous cartilage. Its fibers rupture or become useless for the stability of the disc.

The annulus fibrosus appears to be often intact primarily and to be torn secondarily by the impact of the sharp margins of the fractured bone. Hence, the situation seems to be the reverse of what happens in the herniated disc. Here, lamellae and annulus are torn primarily, and disc material becomes extruded; there the disc material becomes necrotic after fracture of the epiphyseal plate and subsequent overextension of disc fibers. This may or may not be followed by rupture of annulus fibrosus.

These pathological findings suggest the usefulness of a more radical surgical procedure. It may be advisable to remove not only the broken-off fragments of bone and cartilage but to smooth down the remaining sharp bone spicules and to scrape out semidetached particles from the intervertebral space. This is the only condition in which filling of the cavity with bone chips seems reasonable since here is a freshened bone surface to which it may become attached.

If the disc has slipped without rupture of the annulus fibrosus, in other words, if the surgeon has to incise the annulus in order to remove the fragments, it may be difficult to decide during operation which type of injury is present. The collapse of the upper or lower posterior angle of the vertebral body or fracture of the epiphyseal plate may be evident in the x-ray picture so that the surgeon is prepared for what to expect in operation. Lateral, various oblique views and stereoscopic pictures may be necessary to visualize the damage in some instances (Fig. 14). Wedging of the whole vertebra reducing the posterior part of the fifth lumbar to only three-quarters of its anterior half (17 mm. to 21 mm.) is apparent in others (Fig. 15).

I have confined myself in this paper to a description of the pathological changes of the intervertebral disc and its bony attachment in the sciatic syndrome. However, it may be expected that the elastic ligaments which contribute to the stabilization of the spine would become involved by a trauma or in disease independently of the disc, which condition would lead to temporary or permanent impingement upon a posterior root. Elsberg, Spurling, Mayfield and Rogers, Craig and Walsh, Dockerty and Love have pointed to this possibility. This material will be discussed separately since its surgical implications differ from those here described.

SUMMARY

1. Anatomical and pathological investigations of 169 discs removed in operation and 20 controls indicate that the mechanism of the intervertebral disc protrusion is not uniform.

2. Three conditions are described which differ widely in their mechanism and their surgical prognosis. They are:

a. The bulging disc without a rent in the annulus fibrosis and without detachment of

the disc from the bone. Material removed by incision of the disc revealed normal cartilage lamellae.

b. The herniated disc. Traumatized disc lamellae, necrotic or liquefied, are protruded through a rent of the annulus fibrosis into the spinal cavity where they may encroach upon and overextend a posterior root. Herniation of the nucleus pulposus is the exception rather than the rule.

c. The slipped disc in which the trauma involves the cartilaginous epiphyseal plate and which is sometimes accompanied by a chip fracture of the vertebral rim. This frees the disc from its anchorage and permits an eccentric portion of its posterior circumference to slip backward. A posterior root may become hooked up over the protrusion. The sharp bony edges of the fracture may secondarily rupture the slipped disc. It is explained that an individual constitutional disposition may be responsible for this type of lesion. This condition is visible in the x-ray picture.

3. No indication of primary disease of the disc could be detected. Trauma appears to be the only cause of herniation or slipping of the disc.

4. The results of incision and dismemberment of a bulging disc are so poor as to suggest that in the 66 per cent of these patients not benefited by operation, the bulging disc was not the cause of the sciatic syndrome. The inference is that the bulging disc should not be removed unless it clearly involves a root.

5. The results of simple removal of the herniated disc are so satisfactory that there seems to be no need for further improvement of the operative technique.

6. Operation on the slipped disc gives good to fair results in about one-half of the persons operated upon, which fact suggests that there is still room for improvement.

7. The question is discussed under which conditions thorough eventration of the disc may be indicated and when filling of the intervertebral space with bone chips or spinal fusion may be useful.

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REPAIR OF INGUINAL HERNIA WITH EXTERNAL OBLIQUE FASCIA

Critical Evaluation of Results

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INGUINAL hernia and measures for the correction of this anatomic abnormality have long attracted the interest of surgeons of all countries, as evidenced by the voluminous literature on this subject and the many methods of treatment that have been used. It has been widely accepted that an indirect inguinal hernia can be repaired accurately and that a satisfactory result will be obtained. The cardinal features of such repair are high ligation of the hernial sac and reinforcement of both the external and internal inguinal rings. The importance of several other factors has been stressed repeatedly. These factors include intelligent preoperative preparation of the patient as a whole and of the skin of the groin in particular, safe and satisfactory anesthesia, adequate exposure of the structures of the inguinal canal, meticulously complete hemostasis, careful aseptic technique, and accurate apposition of fascia to fascia without tension. It might be thought that attention to these details would lead to permanent correction of the hernia in every case.

RECURRENCE OF HERNIA AFTER HERNIORRHAPHY

On consideration of the incidence of recurrence of hernia after herniorrhaphy, as reported in the literature, one is forced to conclude that there is no perfect surgeon and no perfect surgical procedure. Although many surgeons have gained wide experience in the surgical repair of hernias, when the results of treatment have been carefully tabulated and analyzed in the various medical journals the recurrence rates remain too high. Attention has long been directed to measures which might improve these results.

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A review of the literature indicates that many causes for surgical failure have been advanced. Although Marcy, Halsted, McArthur and others, who wrote a half century ago when herniorrhaphy was first established anatomically as a satisfactory procedure, found that wound infection was the most important etiologic factor in the recurrence of a hernia, sepsis has rarely been incriminated as a major factor in recent years. Bloodgood estimated that 5 per cent of men have a weak or obliterated conjoined tendon, which can be detected by physical examination. He found that the hernias occurred after herniorrhaphy in 50 per cent of cases in which the conjoined tendon was of this type. This anatomic aspect of the problem has been clarified by Anson and McVay (1, 10) who found that in 97 per cent of cases there is a red muscle border at the lateral free margin of the internal oblique muscle which is unsuited for fascia-to-fascia suture to pelvic ligaments and that the tendon of the transversus abdominis muscle (usually tendinous in its lower lateral portion) is inserted into Cooper's ligament on the superior pubic ramus. They further pointed out that reuniting the transversalis fascia, that is, the fascia or tendon of the transversus abdominis muscle, to Cooper's ligament also can be used to repair a femoral hernia or to prevent the development of such a hernia. Burton has reviewed the clinical application of these anatomic findings.

Many surgeons have found that the incidence of recurrent hernia is likely to be high if the original hernia has been present for a long time and if the patient has poor anatomic structures, that is, weak or deficient fascial layers. This is particularly true if the hernial sac is large or if the hernia has extended into the scrotum. In cases in which a truss has

been worn for a long time, the pressure exerted by the truss has been said to have caused thinning and devitalization of the inguinal tissues (8). The increasing incidence of recurrent hernia among persons of advanced age has been noted by many surgeons.

The choice of suture material has been a matter of considerable controversy. Kangaroo tendon and ox fascia have largely been replaced by catgut in herniorrhaphy. In a study of a series of cases in which the patients were followed for from 1 to 10 years after herniorrhaphy was performed, Parsons found that the hernia recurred in 3.5 per cent of the cases in which silk was used as the suture material and in 12.7 per cent of the cases in which catgut was used. In other words, in the cases in which catgut was used, the incidence of recurrent hernia was approximately four times as great as it was in the cases in which silk was used. Cotton, silver and steel wire have their advocates. Many surgeons have strongly urged the use of the types of fascial sutures employed by McArthur and Gallie, respectively. It has been suggested that, if fascial sutures are an added safeguard in cases of large hernias, hernias that are difficult to repair or recurrent hernias, they should be employed in all cases of hernia in order to reinforce the repaired anatomic structures.

Fallis, Zimmerman and other authors have concluded that the choice of the operative procedure in a given case is of minor importance as compared with the selection of the surgeon. Patrick, in an investigation of the causes of recurrence of hernia in the British Army, found that in 63 per cent of the cases of recurrent hernia the hernial sac had not been treated adequately at the time of operation. Fallis has stated that all hernias can be repaired and that all recurrences, therefore, are the result of technical error. It is the surgeon's responsibility not only to avoid errors in technique but also to fit the surgical procedure to the individual patient.

The site of recurrence is a matter on which there is not general agreement. Whether all herniations developing after an attempt at repair of a hernia previously present in the same area should be considered to be recurrent hernias rather than primary hernias is a moot question. Most statistical studies indicate that 75 per cent of primary inguinal hernias in males are indirect and that the remaining 25 per cent are direct in type. The great majority (about 90 per cent) of hernias which occur after repair of indirect or direct inguinal hernias are direct in type and occur low in the operative scar. A recurrence in the vicinity of the internal ring is probably to be interpreted as a technical failure at high ligation and fascial approximation about the cord at the upper end of the inguinal canal. Of special significance in this connection is the report by Grace and Johnson of a follow-up study in 1,032 cases in which operation for hernia had been performed on patients who were more than 50 years of age. In at least 50 per cent of the cases in which the hernia recurred after the operation, the patients were unaware of the recurrence. This finding should be considered in comparing the results reported by different surgeons. For statistical purposes, any hernia which appears in the same groin at any time after a herniorrhaphy must be classified as a recurrent hernia.

The time that may elapse between a herniorrhaphy and the recurrence of a hernia has not been determined definitely. In many of the cases reported in the literature, the patients have been followed for only 1 or 2 years. Statistics based on cases in which the patients have been followed for this period of time suggest that 70 to 80 per cent of recurrent hernias appear in the first year after the operation. Bloch reviewed the results obtained in a large series of cases in which operations had been performed in German clinics. He estimated that 34.8 per cent of recurrent hernias do not appear until 2 years after the operation. Burdick, Gillespie and Higinbotham found that 113, or approximately 40 per cent, of 284 recurrent hernias did not become evident until more than 2 years after herniorrhaphy.

Obviously, an analysis based on the observation of patients for 1 or 2 years after herniorrhaphy has been performed will fail to indicate the incidence of recurrent hernia. It is our opinion that in cases of hernia, as in cases of malignant lesions, the results of surgical treatment cannot be evaluated until at

least 5 years have elapsed since the operation. A review of the literature has failed to reveal any report of a large series of cases of hernia in which the patients have been observed this long after herniorrhaphy has been performed.

THE McARTHUR OPERATION

In 1901, McArthur described a technique for the surgical repair of inguinal hernia by utilizing strips of external oblique fascia as suture material. He pointed out that the permanence of such a repair does not depend on foreign suture material and on apposition of scar tissue but on a lacing of the structures of the inguinal canal with living fascial sutures. A strip of fascia about 4 to 6 inches (10 to 15 cm.) long and about ⅛ to ¼ inch (0.5 cm.) wide is obtained from the medial border of the external oblique fascia. This strip of fascia should not be separated from its attachment to the pubic tubercle. It is used to fasten the conjoined tendon to the inguinal ligament (Poupart's ligament) in order to obliterate the inguinal canal. In cases in which there is considerable relaxation of the tissues, a similar strip of fascia obtained from the lateral border of the external oblique muscle may be used.

By means of animal investigation, McArthur demonstrated that living fascial sutures, that is, fascial sutures that are left attached to the pubic tubercle as previously described, will survive for months or years. This observation was confirmed by postmortem examination of human beings who had undergone herniorrhaphy in which living fascial sutures had been employed.

At present, there is no operation that can be used satisfactorily in all cases of inguinal hernia. An ideal operation would be one that would be followed by a smooth convalescence, satisfactory healing of the incision and a low incidence of recurrence. The McArthur type of herniorrhaphy appears to meet these requirements as well as does any other type of operation.

MATERIAL

In a period of 12½ years, from July 1, 1931, to December 31, 1942, inclusive, one of us (C.W.M.) operated on 332 patients for inguinal hernia. In the early part of this period, ox fascia and catgut were used as suture material in 102 cases. Nylon sutures were used in 8 cases. A modification of McArthur's herniorrhaphy was used in the 222 remaining cases which form the basis of this report. The ages of the patients ranged from 11 to 73 years, and the average age was 47 years. Five of the patients were women and the remainder were males. The occupation of the patients varied widely.

TABLE I.—TYPE OF UNILATERAL HERNIA FOUND AT OPERATION IN 168 CASES

Side of involvement	Type of hernia			Total
	Indirect	Direct	Double sac	
Left	44	8	9	61
Right	86	14	7	107
Total	130	22	16	168

The hernia was classified as a recurrent hernia in 5 of the 222 cases. In 1 of the 5 cases, a hernia had recurred in each groin. The primary hernias were unilateral in 168 cases and bilateral in 49 cases. Table I shows the side of involvement and the type of unilateral hernia encountered in the 168 cases. Bilateral herniorrhaphy was performed in 38 of the 49 cases in which a primary hernia was present in each groin.

SURGICAL TREATMENT

In all of the 222 cases, the patients were anesthetized with nitrous oxide-oxygen and ether. As previously stated, a modification of McArthur's herniorrhaphy was performed in all of these cases. This operative procedure has been used for many years by different surgeons. The technique is briefly as follows.

An incision about 4 inches (10 cm.) long is made nearly parallel to and about ½ inch (1 cm.) above the inguinal ligament (Poupart's ligament). The fascia of the external oblique muscle is divided in the direction of its fibers. The skin is retracted superolaterally to the upper limit of the tendinous portion of the external oblique muscle. A pedicled suture is obtained from the medial border of the external oblique muscle just as it is in the McArthur operation. The fascial suture, which still is attached to the pelvic tubercle,

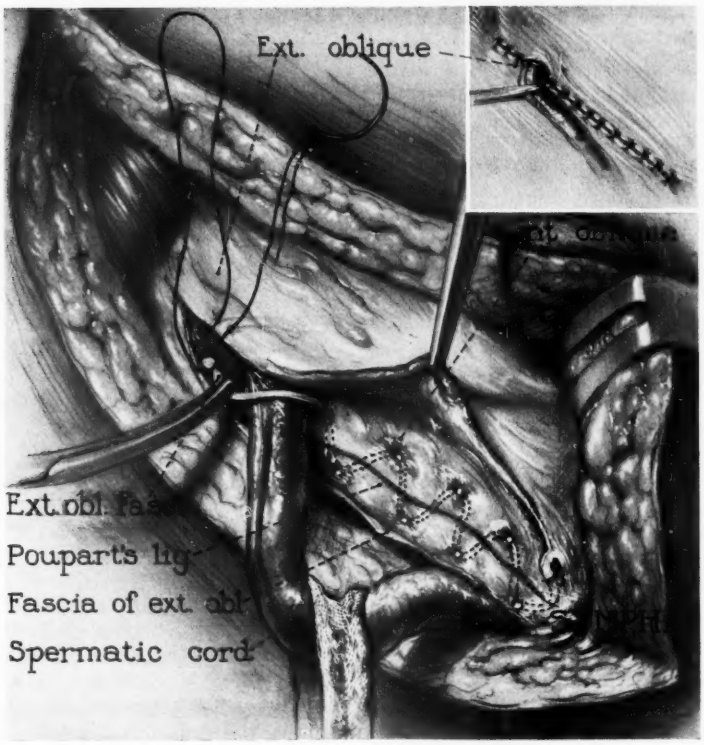

Fig. 1. Technique of herniorrhaphy (modified from McArthur).

is protected with a sponge that has been moistened with a warm, isotonic solution of sodium chloride.

The hernial sac then is ligated as high as possible. If the hernia is of the direct type, the hernial sac need not be excised; instead, it can be infolded satisfactorily with pursestring sutures. The free end of the fascial suture is threaded through a Gallie needle and is used to fasten the conjoined tendon to the shelving edge of the inguinal ligament so as to obliterate the inguinal canal. Although the inferior portion of the free margin of the internal oblique muscle frequently is composed of red muscle fibers, a considerable amount of fascia usually can be found superiorly. This fascia is approximated to the inguinal ligament by rolling the border of the muscle posteriorly. The first stitch that is taken with the fascial suture should penetrate the periosteum of the pubis (Fig. 1) in order to prevent bulging in the lower end of the incision. The external oblique fascia is closed beneath the spermatic cord with interrupted cotton sutures so as to make a new subcutaneous inguinal ring (external abdominal ring) superficial to the abdominal inguinal ring (internal abdominal ring). The skin is closed over the structures of the spermatic cord.

If the patient is a male, a suspensory bandage is applied before he leaves the operating room. The patients routinely are permitted to get out of bed in from 2 to 4 days after the operation, and they generally are dismissed from the hospital before the twelfth postoperative day. At the time of their dismissal, the patients are warned that the hernia is likely to recur if they do any excessive straining within 6 months. They also are advised to do everything possible to prevent the development of

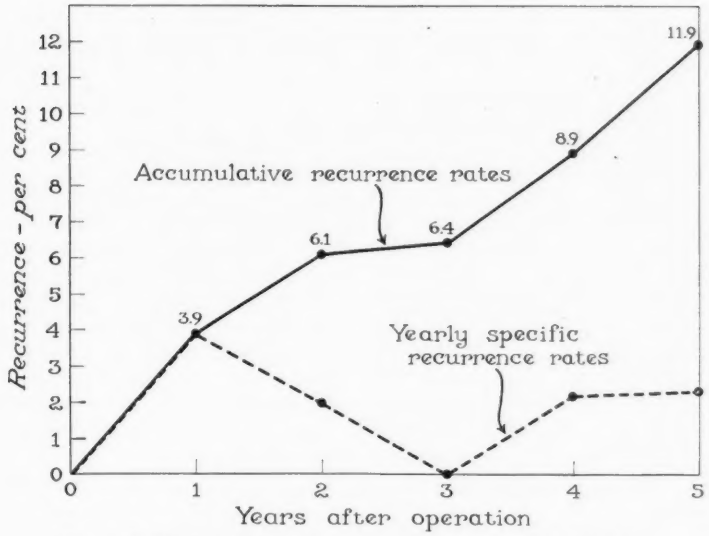

Fig. 2. Incidence and time of appearance of recurrent inguinal hernias.

infection of the respiratory tract and the attendant coughing and sneezing.

RESULTS

Follow-up data were obtained in 208 (94 per cent) of the 222 cases. Since a large proportion of the patients lived a considerable distance from the clinic, the follow-up data were obtained by sending a questionnaire to all the patients who survived the operation. In 2 of the 168 cases in which the hernias were unilateral, the patients died in the hospital within 2 weeks after the operation. In these 2 cases, the patients were men who were 52 and 64 years of age respectively. In each case, necropsy revealed that death was due to pulmonary embolism. Follow-up data were obtained in 155 of the 166 remaining cases.

The incidence of recurrent hernia in this group of 155 cases is shown in Table II. The incidence of recurrence and the time that elapsed between the herniorrhaphy and the appearance of the recurrent hernia are shown graphically in Figure 2. The graph refutes the widely held opinion that recurrent hernias usually appear within the first year or two after herniorrhaphy has been performed. It will be noted that the rate of recurrence was 3.9 per cent in the first year after operation was performed. It is our opinion that the true rate of recurrence cannot be determined until 5 years have elapsed. In this series of cases, the correct rate of recurrence was not 3.9 per cent but 11.9 per cent (Table II).

In 32 cases in which follow-up data failed to reveal any evidence of recurrence of hernia within the first 5 years after operation, similar data also were obtained after 10 or more years had elapsed. In this subsequent period of 5 or more years, a recurrent hernia appeared in only 1 of the 32 cases. In this case, the patient was a man who was 54 years of age when the herniorrhaphy was performed. Although

TABLE II.—INCIDENCE OF RECURRENCE OF UNILATERAL INGUINAL HERNIA

Years after operation	Cases*	Recurrences	
		Number	Per cent
0–1	155	6	3.9
0–2	148	9	6.1
0–3	141	9	6.4
0–4	135	12	8.9
0–5	118	14	11.9

*Inquiry as of January 1, 1947. Table includes only cases in which patients were traced for the specified period.

the result appeared to be satisfactory when the patient was examined 9 months after the operation, bulging appeared in the inguinal region 10 years later. The information obtained by this follow-up study of 32 patients for an additional period of 5 years indicates that 5 years may be considered an adequate period of observation.

Bilateral inguinal hernias were present in 49 of the 222 cases. In 38 of the 49 cases, herniorrhaphy was performed on both sides simultaneously. One of the patients, a man aged 58 years, died of pulmonary embolism on the sixth postoperative day. Follow-up data were obtained in 34 of the 37 remaining cases. In these 34 cases, the recurrence rate was 13 per cent in 5 years. This figure includes every recurrence in this group, regardless of whether the recurrence was unilateral or bilateral. Statistically, this result appears to have been better than might have been expected in view of the recurrence rate of 11.9 per cent in the cases of unilateral hernia. In 11 of the 49 cases in which bilateral hernias were present, herniorrhaphy was performed on one side only.

Operation was performed for a recurrent inguinal hernia in 4 of the 222 cases. In these cases, the results were recorded as satisfactory 9 months, 5 years, 8 years and 11 years, respectively, after the operations were performed. Previous herniorrhaphy seldom distorts the tissues sufficiently to make the subsequent performance of a McArthur type of herniorrhaphy inadvisable.

In the cases in which the patients had worn a truss before the operation was performed, the rate of recurrence was three or four times as great as it was in the cases in which a truss had not been worn. This finding confirms the widely held opinion that special consideration should be given to adequate reinforcement of the inguinal ring in cases in which a truss has been worn. We also found that there was a general correlation between the rate of recurrence and the length of time that a truss had been worn.

Our study did not reveal any significant difference between the results obtained in cases in which the hernia had extended into the scrotum and the results in cases in which the hernia did not extend into the scrotum.

SUMMARY

A review of the literature indicates that widespread confusion exists regarding the cause, site, time of appearance, and frequency of recurrent inguinal hernias. This paper is based primarily on an analysis of the results obtained in 222 cases of inguinal hernia in which a modification of McArthur's operation was performed by one of us (C.W.M.). In 155 cases of unilateral hernia, the recurrence rate was 11.9 per cent in 5 years. In 34 cases of bilateral hernia in which herniorrhaphy was performed simultaneously on both sides, the recurrence rate was 13 per cent in 5 years. The last figure includes all recurrent hernias, whether unilateral or bilateral.

We believe that the end-results of herniorrhaphy cannot be evaluated accurately until 5 years have elapsed since the operation. It is our opinion that the reporting of end-results observed within a shorter period is an inaccurate and misleading practice which should be abandoned.

Although one surgical procedure should not be used routinely in the repair of inguinal hernia, we have found that the use of external oblique fascia by a modification of McArthur's technique is superior to any other method that we have used.

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SURGICAL TREATMENT OF RADIATION BURNS

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EXCESSIVE exposure to x-rays produces changes in the skin and other tissues that sometimes require excision and repair. Radium and other radioactive substances also may cause severe changes; atomic warfare may produce lesions similar to those seen in local areas at present. The skins of some individuals may be unusually sensitive to the x-ray and exposure may produce acute episodes that may clear up in time. Permanent chronic changes, however, develop in practically all skins which have been exposed to heavy or repeated excessive or incorrect dosages of x-rays.

These comments have no relation to the usefulness of, or indications for, x-ray therapy, nor are they meant to suggest any directional approach to the problems in the use of radiation therapy.

SOURCES

Burned hands of physicians are the most frequently seen at the present time (Fig. 2). Many of these burns occur while difficult fractures are being reduced under the fluoroscope; the physician's anxiety for the welfare of his patient may have dulled his regard for his own personal safety. Nevertheless, such burns are preventable and every physician using an x-ray machine should be aware of the effects of a single large dose and of the irreversible, cumulative effect of repeated small doses over many years.

The list of early workers in radiation who have been burned is long and contains many names well known to the profession. Dr. Walter Cannon, late professor of physiology at Harvard, suffered burns of his hands that required excision and grafting. His burns resulted from exposure while doing pioneer work in his x-ray studies of the gastrointestinal system, before the late effects on the skin were known.

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Dentist's fingers are sometimes burned (Fig. 3), especially if they hold films in the mouths of patients during exposure.

Prolonged fluoroscopic examinations have produced some most unfortunate burns as a result of miscalculations in distance, time, or filters (Figs. 1, 15, 16, 20, 21). Examinations of empyema fluid levels, fractured metatarsals, and fractured vertebrae have produced especially bad burns. As these burns are of such great trouble both to the patient and to the one responsible for them, it seems advisable to give warning of this source.

Acne, eczema, port-wine stains, plantar warts, epidermophytosis, and pruritus ani are all troublesome diseases, but are as nothing compared to the possible effects of excessive radiation therapy (Figs. 4, 8, 9, 11, 13, 14).

"Sailor's skin," or "farmer's skin,"—the dry, atrophic, telangiectatic skin resulting from decades of heavy exposure to sunlight—is especially intolerant of x-radiation. The pathological changes in this type of skin are similar to those in a mild x-ray burn, and it is probably advisable to treat by other means keratoses, skin cancers, and even lip cancers in such patients.

Commercial epilation of superfluous hair by x-ray has caused some of the most dramatic, extensive, and useless burns (Figs. 5, 17, 18); patients have been seen with burns of the entire lower legs, the outside of the thighs, the axillae, outside of upper arms, entire forearms, hands, back of neck, entire face, and entire front of neck. The prolonged morbidity, suffering, and loss of economic and social status endured by these patients cannot be condoned in the face of the slightness of their original complaint of superfluous hair. This commercial procedure cannot be too severely condemned.

Hemangioma and plantar warts. As more time elapses the distant effects of radiation treatment will become more apparent, and perhaps in some instances, changes in therapy

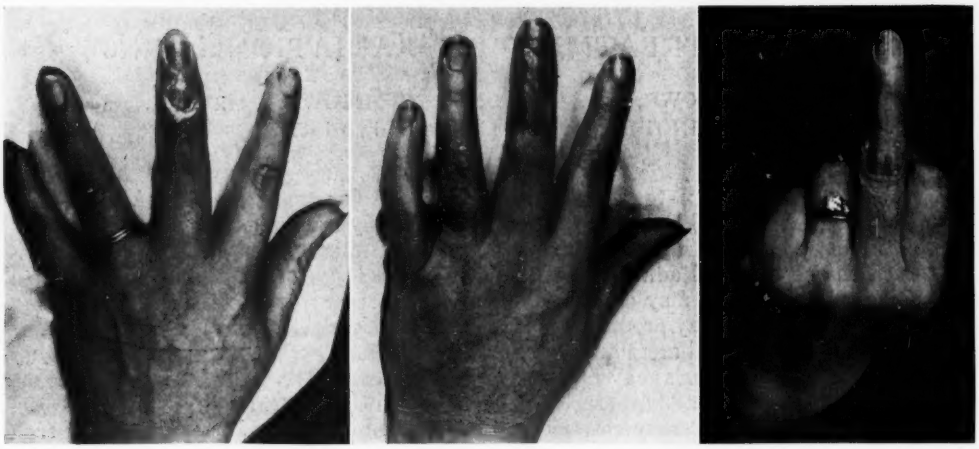


Fig. 1. Fluoroscopic burn following exposure to locate foreign body. Complete early resection with immediate repair with split graft. One operation, normal function.

or dosage will be instituted. Typical lesions that are treated early and in which later dermatitis and chronic atrophy may result are hemangiomas, especially about the face (Figs. 9, 10), and plantar warts. Radiation lesions of the sole of the foot are especially difficult to treat; even cross-leg flaps may be necessary for repair (Figs. 15, 16).

Fig. 2. Fluoroscopic burn of doctor. a, b, Acute burns on both hands—left worse than right. Patient required six grains of morphine a day. Patient came in with consideration of amputation of hand. c, d, Result of general protective care and early resection and free grafting. Recovery was such that he was able to follow a full career in anesthesia whereas he formerly was a surgeon.

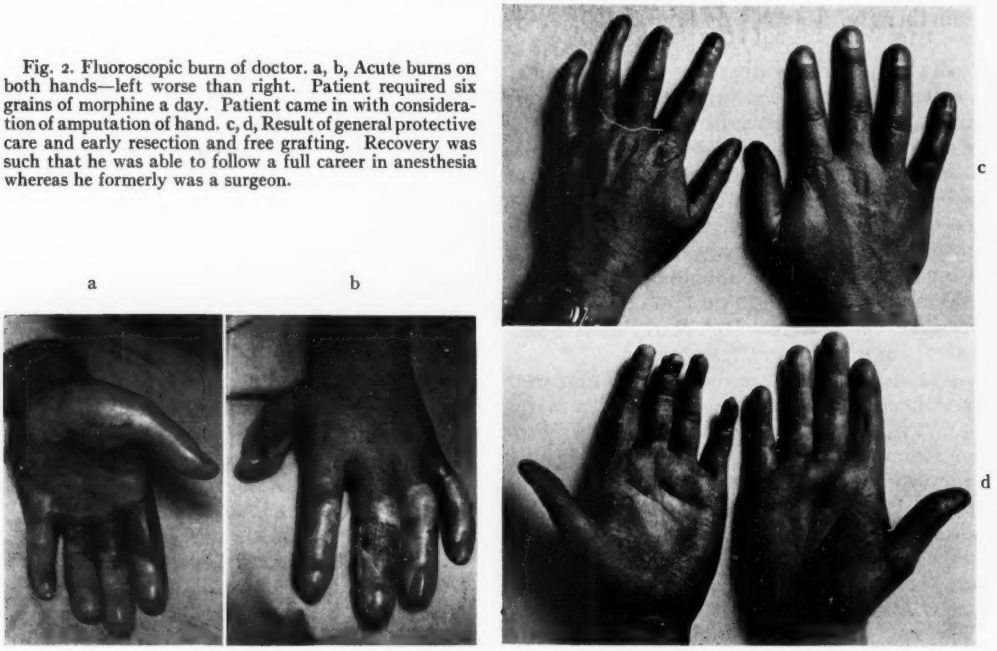


Fig. 2.

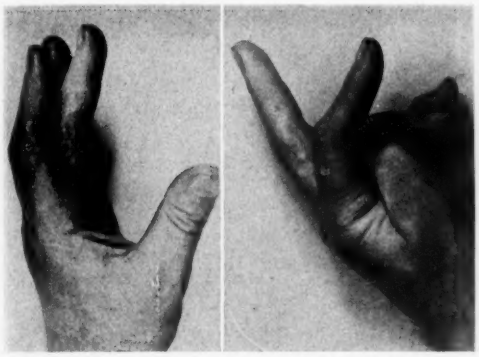


Fig. 3. Dentist's burns. Typical burn of dentist's fingers from holding films in patients' mouths. Resection with immediate split graft. One operation.

Fig. 4. Eczema burn. Wide deep burns of both hands from treatment for eczema. Repair by resection and replacement of most of the skin of both palmar surfaces including fingers. Two operations were necessary. Function was about normal. To have additional work on fingers of right hand.

It is hoped that none of these statements will be interpreted as underestimating the very great value of radiation therapy and x-ray examination for many conditions; the x-ray is invaluable and indispensable. Recognition and elimination of the unfortunate ultimate results here discussed may lead to eventual wider usefulness of the various radiation procedures.

PATHOGENESIS

Wolbach reported the essential pathological processes which include:

"1. Loss of appendages of the skin.

"2. Replacement of normal collagen by a peculiar dense hyaline collagen rich in elastic fibers and poor in cells.

"3. Obliterative processes in blood vessels of the corium and subcutaneous tissues.

"4. Necroses of varying sizes in the corium immediately beneath the epidermis. In the earlier stages these are usually in the region of thrombosed telangiectases. In the later stages telangiectasis may be nearly entirely absent.

"5. In response to necroses of the corium, reparative proliferation on the part of the epidermis," which may extend beneath the telangiectasis or areas of necrosis. "These small necroses containing the thrombosed telangiectases become completely separated from the skin by the layer of regenerated epithelium and persist for a time as dry, dark-colored spots—the 'coal spots'—until cast off. The significance of these processes lies in the fact that they are invariably present in chronic x-ray dermatosis even after the lapse of many months or several years since the last exposure to radiation." Malignant properties may be acquired after years of excessive downward proliferation that is called forth by the changes which take place in the corium and subcutaneous tissues.

The blood vessel changes may be of extreme importance. Early, there is a marked thickening of the walls of the arterioles and venules at the junction of the dermis and subcutaneous fat (Fig. 6a). The occlusive endophlebitis seems often to be more severe than the endarteritis, resulting in back pressure on the subepithelial capillaries with dilatation and telangiectasis (Fig. 6b). Stagnation of

Fig. 5. Commercial hair remover burn. Excessive burning from commercial x-ray treatment to remove hair. Patient's arms, axillae, and legs were completely burned. Treatment consisted of excision, in two stages, of all skin of lower arm and immediate split grafting.

the blood flow in these dilated capillaries is followed by thromboses, which later become organized and in the process may rupture the vessel walls with some leakage of blood cells into the subepithelial spaces. These black "coal spots" are grossly visible and are usually several millimeters in diameter. It is well known that the dermis is intolerant of macroscopic foreign bodies (for example, fine silk sutures will remain indefinitely in the subcutaneous tissues but they are always cast out of the dermis).

Thus the development of carcinoma in radiation lesions may be about the closest that we have come to the actual knowledge of the formation of carcinoma, clinically. The epithelium is continually stimulated to downward growth for so many years to cast off the recurring "coal spots" (Fig. 6c) and to heal the recurring ulcerations that it finally loses the normal growth response (to these foreign body and wound stimuli) and just goes on to malignant change (Fig. 7).

The clinical stages of atrophy, telangiectasis, "coal spots," keratosis, and carcinoma are typical for chronic burns, such as those found on physician's hands or such as follow repeated treatments given for acne, goiter, or excessive hair. The progress of the stages is different for different patients and different exposures, but generally there is telangiectasis in from 3 to 10 years; "coal spots" and keratosis in 10 to 15 years; and carcinoma in 20 to 25 years. Malignant change has been noted as early as 3 years, however.

An interesting thing is that in the donor site from which a split graft is taken to repair an x-ray burn, the opposite reaction takes place in that the deep epithelial cells de-differentiate to re-cover the bare surface that is left when the skin is split in two.

The carcinomas that develop in radiation burns are usually squamous cell in type, though occasional basal cell or basal squamous lesions are seen. The squamous cell lesions can and do metastasize, while the basal cell lesions often show "rodent ulcer" characteristics, with deep local invasion.

X-ray burns are usually heavily contaminated with a wide variety of virulent bacteria. This is due partly to the many tiny crypts and recesses afforded by keratoses, recurrent ulcerations, and cast-off "coal spots," and partly to the cessation of sweat gland and sebaceous activity with loss of the normal skin-cleaning mechanism. The lesions may be so painful

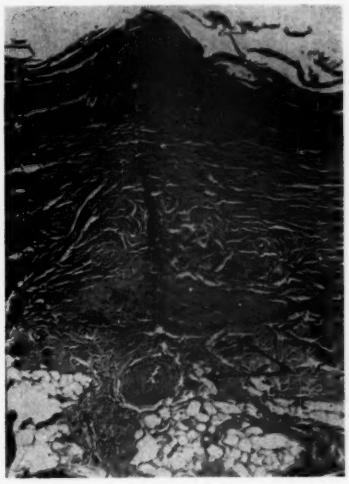


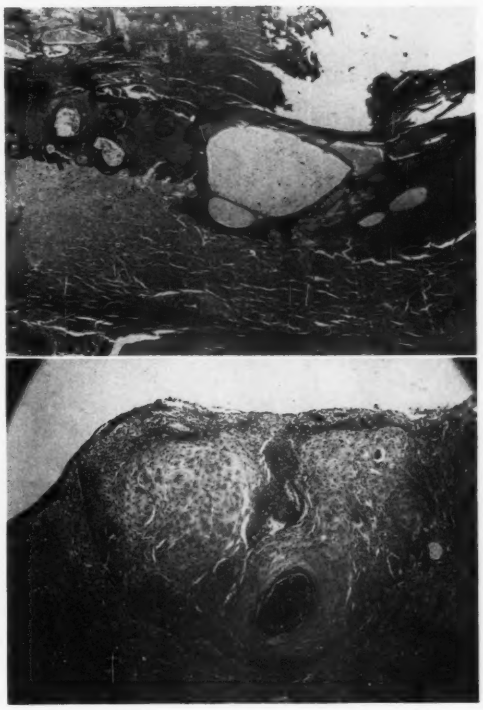
Fig. 6a

Fig. 6. a, Shows endarteritis and endophlebitis at junction of skin and subcutaneous tissue, with complete occlusion of venule and almost complete occlusion of arteriole. Loss of skin appendages. b, Telangiectasis of subepithelial capillaries, probably as result of obliterative changes of venules draining skin. c, Typical "coal spot." Dilated thrombosed telangiectasis with surface epithelium beginning to grow down around it to cast it out. Hyperkeratosis over the area.

Fig. 7. a, Casting off of numerous "coal spots." The epithelium is shown actively growing "under coal spots" (clotted vessels) in the process of casting them off. Excessive hyperkeratosis probably as a product of the short life cycle of the cells, which may be brought about by the continual and excessive wound stimulus of the "foreign bodies" in the dermis. In the upper left can be seen clotted vessels in the keratin that have already been cast out. b, Development of carcinoma from the continual wound stimulus. The epithelium apparently finally invading in general, instead of purposefully, to get rid of "coal spots."

b, above c, below

Fig. 6.



a, above b, below

Fig. 7.

Fig. 8. Widespread involvement following treatment for acne. As usual the tip of the nose being closest to the tube is the worst burned. Repair by resection and immediate split grafting. Four operations were necessary to repair both cheeks, whole nose, upper lip.

Fig. 9. Early occurrence of burn following radium treatment of hemangioma. Repair by excision and full thickness graft from clavicular region. One operation.

that they are left grossly dirty because the patient simply cannot tolerate the added pain of having the wounds cleaned. This cleansing is an important factor in the consideration of any surgical treatment, and its omission was responsible for some grave wound infections before the days of effective chemotherapy. A corollary to this is that these wounds and areas will not tolerate the local application of strong antiseptics.

TREATMENT OF ACUTE BURNS

Acute burns are usually the result of a single massive dose, often with inadequate or no filtration. Such burns are most commonly sustained after fluoroscopy, either upon the hands

Fig. 10. Atrophy and deformity following radon treatment of hemangioma. Excision and repair with cross lip flap. Two operations.

Fig. 11. Eczema burn. Extreme involvement, with malignant loss of nose and chin and with multiple carcinomas throughout areas. Repair by complete excision and immediate free grafting. Four operations. Prosthetic acrylic nose made by Miss Gertrude Hance.

of the doctor (Fig. 2) or in patients who have had elusive foreign bodies buried in the tissues.

Even in doctors, the true nature of such burns is sometimes not apparent and so the lesion may be mistakenly treated as a severe allergic condition or as a dermatitis medicamentosa. These areas are apt to be markedly swollen and edematous, reddened, weeping serum, and characterized by excruciating, deep boring, throbbing pain. They typically become worse each day, for a week or two, and seem to be refractory toward all treatment. In the worst cases, the skin may turn white

Fig. 12. Malignant breakdown of an old x-ray burn, in an area treated for tuberculosis of the lymph nodes. Wide deep resection of the carcinoma (done by Dr. Bradford Cannon) and secondary repair with thick split graft. Two operations.

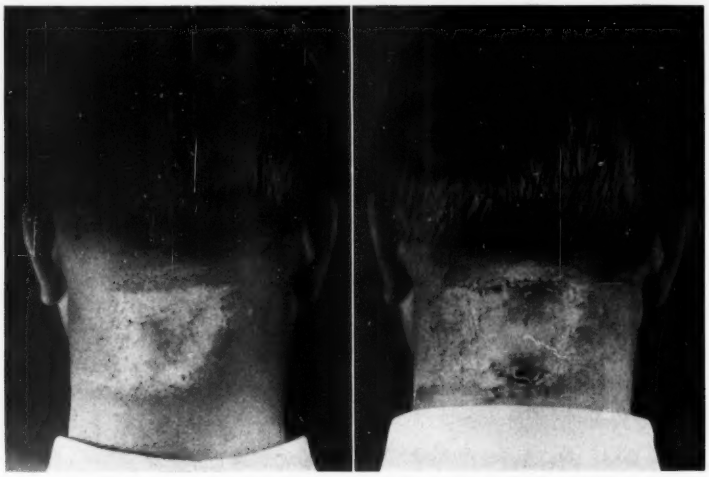


Fig. 13. Acne burn. Widespread burn in a doctor following treatment for acne. Resection and immediate free graft shown after 2 weeks. One operation.

after a few days (as in a deep fire burn) and then slough, leaving a foul, greenish-gray, sloughing ulcer. In the milder burns, the redness, swelling, and pain gradually subside and for a few months, at least, the skin appears fairly normal.

The pain in acute burns may be so severe as to require hospitalization and heavy sedation. One patient required 6 grains of morphine a day, and the tendency here is noteworthy of caution.

As some acutely burned areas recover, or at least persist as adequate cover for the part, and as there is not much chance of determining the extent of damage of an acute burn, very early operation is seldom indicated.

Local anesthetics are usually unavailing and may add a secondary drug dermatitis. The use of aloe vera (an antipruritic which was formerly used for a variety of skin conditions) has been suggested and, if obtainable, may relieve itching and some pain to a worthwhile

Fig. 14. Athlete's foot burn. Extremely crippling x-ray burns following treatment of athlete's foot. Resection of lesion and immediate repair with thick split grafts. Two operations—one over ankle and one over most of heel.

degree. Getting it fresh and keeping the bulky split leaf in place are details that few patients will carry out very long, however.

Burned areas are less painful if they are sealed off from the air by some bland ointment (e.g. cold cream or lanolin, *not autoclaved*) and a loose, soft, cotton dressing without any gauze next to the skin. Elevation and immobilization of hands may be quite helpful. Strong antiseptics, especially those containing heavy metals, are to be avoided. Gentleness, cleanliness, and the avoidance of irritants are the fundamentals necessary for good local treatment.

The use of ultraviolet or further radiation of any kind is definitely not advisable and is mentioned here only because it has been recommended. There seems to be no justification to use further radiation therapy in an effort to relieve such lesions which are in themselves the result of exposure to too much radiation.

If the burn has been from a single cauterizing dose in which the limits are fairly well defined and ulceration seems likely with little chance of adequate healing, early excision and grafting may be worth trying. The depth of the trouble may not be apparent, however, and there may be difficulty in growing a free skin graft.

TREATMENT OF CHRONIC BURNS

The diagnosis of chronic burns is often missed because the patient may be unaware of the cause of his condition. This fact is true even in some physicians who do not associate the chronic dermatitis of their hands with the holding of a fluoroscopic screen for a few minutes each week. The power of recovery of the tissues from the long continued effects of radiation seems to be slight or nil, so that the status of a given individual's skin seems to be determined by the cumulative amount of radiation that he has received throughout his life.

The triad of atrophy, telangiectasis, and keratosis of the skin should lead to the diagnosis. About the only condition that is to be

Fig. 15. Fluoroscopic burn. Extremely painful, completely crippling lesion from one fluoroscopic examination following injury. Involvement clear through onto dorsum. Repaired in two operations by wide excision and split grafting.

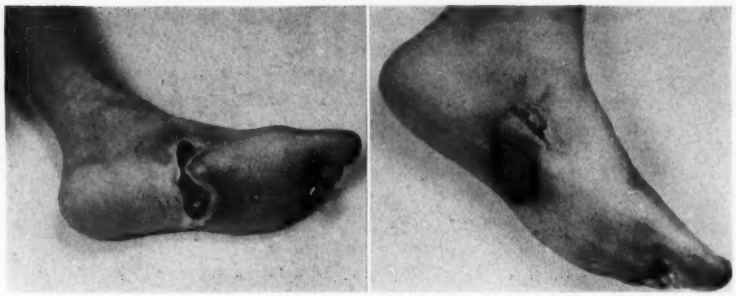


Fig. 16. Sole of foot burn extending so deep that cross leg flap had to be used, rather than free graft. Two operations.

differentiated is "sailor's" or "farmer's skin," which is similar, but is caused by the cumulative effect of decades of overexposure to sunlight in persons who do not form sufficient melanin to protect themselves (in other words, they do not "tan"). Differentiation can be made by the history and by the fact that "sailor's skin" symmetrically affects both forearms, both hands, and the entire face and neck in most patients. Telangiectasis is often predominant in x-ray burns, whereas atrophy and keratosis predominate in "sailor's skin." "Coal spots" rarely occur except in association with x-ray burns. Carcinoma may develop in either.

The progress of a severe chronic x-ray burn through the stages of atrophy, telangiectasis, "coal spots," keratosis, and carcinoma is irreversible and inevitable if the patient lives long enough. This course is not affected by the use of any drugs or other means of treatment known at present except the surgical excision and repair of the area. The stages may overlap and the speed of the progress varies greatly so that it is often a major problem to decide when and if to undertake excision. Fast progress in any patient and even slow progress in a young individual are indications for treatment. A paradox of treatment

Fig. 17. Commercial x-ray treatment for removing hair, resulting in almost total burns of lower legs.

Fig. 18. Same patient as in Figure 17 shown after repair by wide resection and grafting. Surrounding involved area done at subsequent operation.

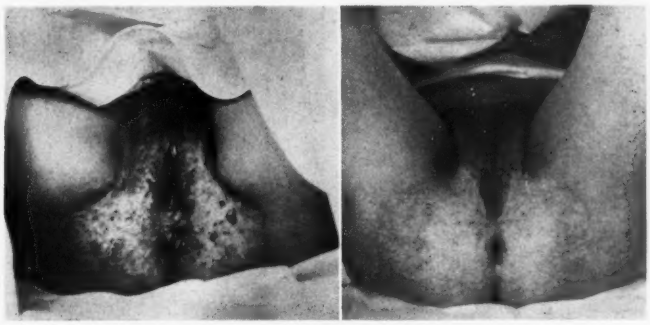


Fig. 19. Pruritis ani treatment. Excessive involvement, repaired by excision and immediate grafting. Two operations.

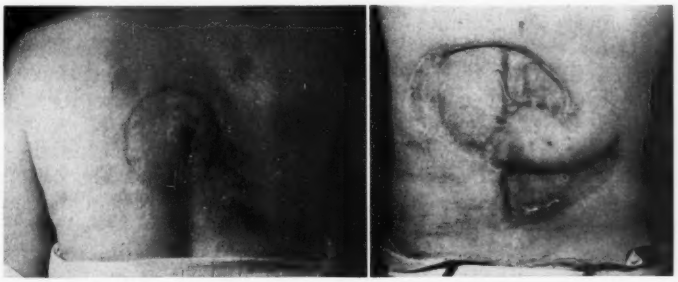


Fig. 20. Fluoroscopic burn. a, left, Wide deep involvement following chest examination. Repaired later in one operation. b, Another patient with similar burn the result of gastrointestinal fluoroscopy, but with a direct flap added to cover a deeper involvement.

is that the excision and repair should be done when the area is in relatively good condition —not waiting until ulceration, pain, and malignant change have occurred. However, many patients wait for repair until these stages have occurred so that the greatest surgical problem lies within this group.

Faint telangiectasis and atrophy may not require treatment if it is very slowly progressive, but even in this group excision and repair are sometimes indicated for cosmetic improvement.

Bland ointments may soften "coal spots" and keratoses and seem to stop them for a time, but the underlying pathological changes are progressive and later become manifest. On hands, keratoses develop with the "coal spot" stage and these may rapidly become malignant. These spots can best be removed in mass and the areas skin-grafted. It is less desirable, but feasible in some patients, to do cautery excisions of the individual keratoses as they occur and let the areas heal by secondary intention. These are the unusual lesions for which more radiation is sometimes recommended, but it should *not* be used as it is apt to accelerate the progress of the pathological changes. For the same reasons, carcinomas in x-ray burns should *not* be treated with more radiation.

When it is decided that a radiated area needs excising, the size, depth, location, ulceration, and malignant change in the area should be carefully considered. The basic therapy is wide, deep excision followed by immediate closure of the wound with the use of free split skin grafts. The following variations in technique are sometimes useful:

1. The wound is left open to be skin-grafted later. This step is taken because these areas

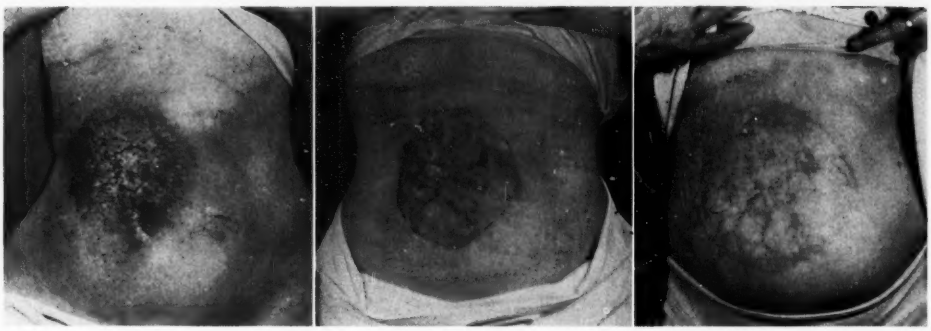


Fig. 21. Abdominal wall x-ray burn following treatment for gastrointestinal tract lesion. Complete excision, immediate grafting. One operation, 8 days in hospital.

are apt not to have a very good minute (capillary) blood supply (even though blood may pour over the whole field from a few arterioles and venules), and with an inadequate minute blood supply, transplanted tissue does not survive.

2. The wound may just be "dressed" in a split graft and the patient advised that the graft may not take and that the graft may have to be repeated if it is suspected that the blood supply is inadequate.

3. Immediate flap closure of the defect either by adjacent flaps or distant flaps prepared ahead of time may be necessary and definite plans should be made for such a contingency.

4. Delayed closure of defects with pedicle flaps may be indicated. This is true when there is no doubt that the area needs a flap, and when the depth of excision is uncertain, as for instance around the vertebrae or in any place in which the adjacent nerve supply must be protected.

5. In the presence of exposed bone or tendons, pedicle grafts should be used. Free grafts do not do well over bone and will not grow directly on bare bone or bare tendon (if the area is more than a few millimeters wide), whereas pedicle flaps will usually survive and successfully attach themselves in such situations.

There are two essentials: (1) to get rid of the lesion, and (2) to repair the area for useful function and appearance.

The persistence of function of skin grafts will be satisfactory if the involved tissue has been excised widely and deeply enough. Edges should be watched and if deep trouble develops, wider and deeper removal and repair may be necessary.

The preparation of wounds for operation consists of local cleansing, the use of mild local antiseptics (such as 1:5,000 aqueous zephiran) and on feet, non-wettable agents (such as some of the silicate-gel preparations) may be used to prevent maceration of the surrounding skin under the wet dressings.

These basic steps for repair are used throughout the body, but some local areas may be considered separately.

Hands. Chronic x-ray burns of the hands occur most often in physicians and dentists and respond well to wide removal of skin and repair with thick split grafts. The heaviest burn may be either on the dorsal or palmar surface, but the opposite surface is nearly always involved to some extent. Excessive pain may occur and require heavy sedation. In lesions of very long duration, the individual blood supply to the fingers may be of such low quality that care has to be exercised in maintaining what there is available—in not doing too radical operations—not sacrificing both arteries of a finger at one time—not getting bandages too tight. When definite carcinoma has developed, very wide deep excision is necessary, but since excision cannot be very deep without sacrificing function amputation must always be considered as possibly the best and safest procedure in some patients. These lesions do not always remain local

they should be examined and watched periodically for possible regional lymph node involvement. One of the main difficulties, of course, is to get the physician to submit to an adequate operation.

Face. Radiation burns of the face carry the added responsibility of securing not only good repair but a good cosmetic result. The paradox here is that radical operation should be done early, before the features have been damaged by the development of carcinoma. At times when patients have been treated for acne or eczema, it is necessary to excise and replace the skin of the entire face—nose, cheeks, lids, forehead, eyebrows, and chin. If excision can be done early enough so that free skin grafts can be used, the functional and cosmetic results are far better.

It has been noticed that the little difference in closeness of the tip of the nose to the x-ray machine usually produces enough more radiation as to cause greater damage here and in addition a resulting chronic mucositis of the nose if not a burn, must be treated.

Feet. Radiation lesions of the sole of the foot form a story of their own; there are frequent histories of years of debility and great difficulty of repair. Prophylaxis is as important here as in any region, for the disability may be extreme and prolonged.

The rules of treatment are the same—excision and repair. Free split skin grafts can be used even for wide areas, if operation is undertaken before ulceration has occurred. Small ulcerations, if not too deep, can often be repaired by local flaps or free grafts, but cross-leg flaps are almost always needed for repair of extensively ulcerated lesions. The idea of cutting into the sole of the opposite foot to secure a graft for the repair of a damaged one is mentioned only because it has been advocated and to suggest that it should never be done.

Fluoroscopic burns of the foot may cause some of the most distressing deep involvements encountered. Where the plantar fascia is involved shreds may be extruded at intervals for months with total disability and considerable pain throughout that time. The deep joint structures as well as the opposite surface of the foot are especially apt to suffer in these foot lesions. The chronicity of the process far outweighs any value of the fluoroscopic examination.

Fig. 22. Hemangioma treated by radium in childhood (elsewhere). Surgical replacement of entire half of face with massive delayed chest flap.

Rectogenital area. Perianal and perivulvar burns (usually from treatment of pruritus) offer their special problems in repairing such contaminated areas and dressing such a difficult field (Fig. 19). Excision and immediate or delayed free grafting, or the immediate shifting of local flaps inward and the grafting of their sites is usually carried out. Possible troubles from a circular scar near the pectinate line should be considered and avoided if at all possible.

Preoperative preparation may include enemas and laxatives to empty the bowel and oral streptomycin or sulfasuxidine to reduce the bacterial flora. Frequent sitz baths may help in preparing the local skin.

At operation, the "stent" type of fixation may be used on grafts, long edge sutures are tied over a pad of gauze to immobilize the graft and dressing and the edges are surrounded with zinc oxide or some other impervious ointment. The use of a retention catheter and low residue diet for 7 to 10 days may help greatly in the prevention of soiling. After that period, cleansing of the grafted area, after each soiling, is carried out with the grafts usually becoming quite stable and requiring little further care after a period of 2 to 3 weeks.

Relief of pain by operation. Pain in these burns may be so severe and so refractory to

sedation as to require immediate wide, deep excision of the lesion, with or without primary repair. When pain is so outstanding, very dramatic relief is usually obtained by excision. The patient, upon awakening from the anesthetic, and in spite of the discomforts of the operation, will volunteer the information that his pain is gone.

Severe pain may occur in lesions on the neck, in areas adjacent to bone, and in lesions along the vertebrae, where root pain may be excessive. One patient with severe abdominal pain, who had a large burn of the back was nearly subjected to a laparotomy, but was completely relieved of his abdominal pain by deep excision of the ulcer on his back (Fig. 20). This is likewise especially true in patients with painful anal lesions.

The pain from excessive radiation about the jaws is one of the most difficult to alleviate.

FINAL REMARKS

These lesions are unhappy ones from every possible aspect, and they are more common than is generally believed. Their prevention should be paramount in all instances of exposure. Their occurrence should be recognized early, and complete excision and repair should be done before malignant changes occur (Fig. 21). Such a regimen will greatly reduce the total amount of disability and suffering in these patients.

It may be repeated that severe lesions are progressive and that surgical treatment should be undertaken before serious ulceration occurs and carcinoma develops.

IMPERFORATE ANUS

A Case Series and Preliminary Report on the One Stage Abdominoperineal Operation

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IMPERFORATE anus was recognized and described in the third century, A. D., but it was not until 1835 that the operation of perineoplasty with mobilization of the rectal pouch was recommended. In 1844, Velpeau advocated sigmoid colostomy following unsuccessful perineal dissection. With the use of these procedures and primary colostomy, the operative mortality has remained essentially unchanged since 1915 (6, 21).

INCIDENCE

Congenital anomalies of the anus and rectum are said to occur in the ratio of 1:5000 to 1:10000 live births. Males are affected somewhat more often than females. This disparity was greater than 2:1 in this series. It is unusual to have similar anomalies in siblings, but this does occur. Two sisters were so affected in the group of cases at hand.

EMBRYOLOGY

The development of the lower rectum, anus, and urogenital tract have been described in detail by other authors (1, 2, 4, 6, 7, 13, 14, and 18). In brief, these structures become differentiated between the fifth and eighth weeks of embryonic life. At first, the allantoic duct and the hindgut form a common cavity, the cloaca, which is then divided by a downgrowth of mesoderm, the urorectal septum, into an anterior portion, the forerunner of bladder and urethra, and a posterior portion, the primitive rectum. The anus is formed by an external invagination, the proctodeum, separated at first from the rectum by the anal membrane, which later ruptures, establishing continuity of the lower gut.

Rectourinary fistulas represent a failure of the urorectal septum completely to divide the cloaca. In the female, the muellerian ducts, the lower portion of which form the uterus and vagina, descend in the urorectal septum, taking over any fistulous connections, if present.

The external anal sphincter develops from the regional mesoderm and therefore smooth muscle is usually present in the perianal area in spite of anomalous anal development. However, the position and function of this muscle are frequently distorted by the anomaly (4).

TYPES OF ANORECTAL ANOMALY

Malformations of the anus and rectum are usually classified into four main types (13, 14) (Fig. 1). In type I there is a stenosis of the anal mucocutaneous junction or of the lower rectum due to incomplete rupture of the anal membrane. No actual atresia is present. Type II is an imperforate anus with a persistent anal membrane only. Type III is an anomaly in which the rectal pouch ends blindly at a variable distance above the perineum. It is thought to be due to excessive degeneration of the postallantoic portion of the hindgut (13). The anal site is often represented by a dimple in the skin. This type is by far the most common (6, 10, 12, 13, 14, 18). In type IV, the anus and lower rectum are normally developed. The upper rectum ends blindly at a variable distance above this distal portion, probably due to concrescence of the superior portion of the rectal ampulla (13).

Fistulas may be present in any one of the four types of anorectal anomaly but are most commonly seen in type III (Fig. 2), least in type IV (6, 12, 13, 14, 18). Multiple fistulas are extremely rare (13, 14). In males, the fistula may be rectovesical, connecting the rectal pouch with the bladder in the vicinity of the trigone; rectourethral, extending from the

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TABLE I.—TYPES OF ANOMALIES

Type of anomaly	Males	Females	Type total	Total fistulas in each type
I	1	1	2	0
II	10	3	13	3
III	25	12	37	26
IV	0	0	0	0
Totals	36	16	52	29

TABLE II.—DISTRIBUTION

Fistula	Type II males	Type II females	Type III males	Type III females	Total fistulas
Rectovesical	0	0	8	0	8
Rectourethral	0	0	6	0	6
Rectovaginal	0	0	0	10	10
Rectoperineal	3	0	1	1	5
Totals	3	0	15	11	29

pouch to the prostatic or membraneous urethra; or rectoperineal, a tract leading from the rectum externally to the perineum anterior to the anal dimple. In females, the fistula may be rectovaginal or rectoperineal. Rectourinary or rectouterine fistulas are very uncommon. High rectovaginal fistula does occur, although most of these tracts open into the lower third of the vagina or into the vulva immediately anterior to the fourchette. "High fistulas" include rectourinary and high rectovaginal connections; lower rectovaginal, rectovulvar, or rectoperineal are termed "low fistulas."

In this series of 52 cases, there were 2 patients with type I anomaly, 13 of type II, and 37 of type III. There was no patient with a type IV defect. A fistula was present in 29 of these cases, or 55.8 per cent (Table I).

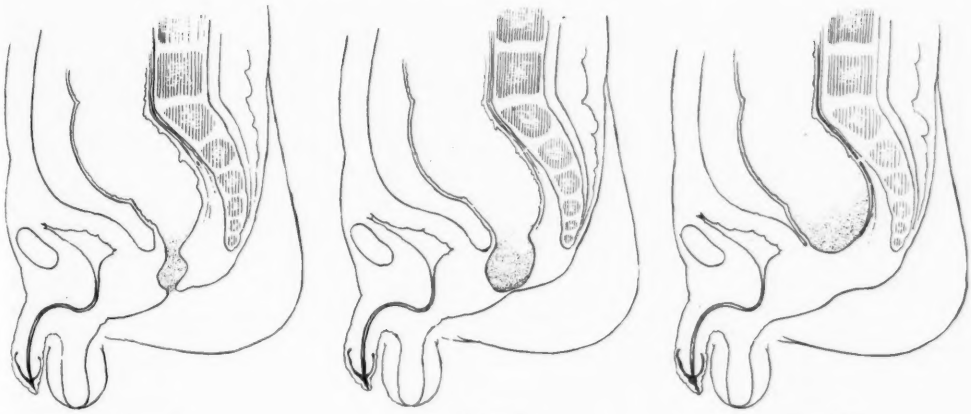
The distribution of various fistulas is shown in Table II. Attention is drawn to the fact that all but 3 of the fistulas occurred in patients with a type III anorectal defect, a fistula incidence of 70.3 per cent in this type.

CLINICAL EVALUATION

In general, patients with congenital anorectal anomalies fall into two main clinical divisions. The larger group consists of type I cases with extreme stenosis, and the other types without a fistula or with a narrow fistula, who enter the hospital with actual or impending intestinal obstruction. The smaller division includes the more common type I case, together with the other types having a low fistula of a size sufficient to pass feces. These enter for correction of the malformation and frequently for relief of chronic partial obstruction with fecal impaction. In this series there were 33 patients of the first or emergency group. The average age of entry was 27.0 hours. There were 12 patients of the second clinical division, varying in age from 6 days to 3 years, averaging 8.3 months on entry. The type I cases were the youngest patients of this group, the type III cases with low rectovaginal fistulas, the oldest. The 7 remaining patients of the series entered the hospital for secondary operations, the primary procedures having been performed elsewhere.

Of the 33 patients in the potentially obstructed group, only 4 showed clinical evidence of actual obstruction. Two of these entered at the age of 48 hours, the others at 60 and 72 hours, respectively. All exhibited abdominal distention in moderate degree, intestinal patterning was present in 1. Seven of the potentially obstructed patients were subjected to laparotomy at ages varying from 3 to 77 hours, and all showed varying degrees of colonic distention at surgery although in only the oldest 2 was clinical abdominal distention noted preoperatively. One-third of the nonemergency group of patients entering at a later date for correction of the anal deformity had chronic partial large intestinal obstruction with fecal impaction palpable abdominally.

The diagnosis of the type of anomaly can be made by inspection and palpation of the anal and urogenital regions (13, 14). A dimple was noted at the anal site in 13, and puckering of anal skin in 2 of 37 cases of type III anomaly. It is felt that one or the other of these signs occurs in a greater percentage than these findings indicate. External fistulas (rectoperineal and low rectovaginal) are readily apparent, but rectourinary and high rectovaginal fistulas often escape detection. A careful preoperative gross and microscopic examination of the urine should be made in all patients in whom no external fistula is apparent. However, a high narrow fistula may be blocked temporarily with inspissated meconium and produce no



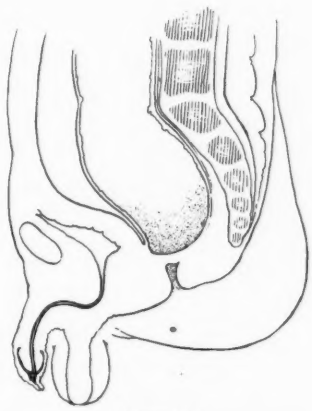


Fig. 1. Diagrammatic midsagittal sections showing types of congenital anorectal anomalies. (Classification of Ladd and Gross.)

immediate evidence of its presence (13, 14). Of 14 rectourinary fistulas (all in males), 7 were not diagnosed prior to operation. Although not all of the primary procedures were performed at this hospital, this represents an overall diagnostic failure of 50.0 per cent in the early determination of rectourinary fistulas. Of 10 rectovaginal fistulas, 2 (both high in the vagina) were not diagnosed preoperatively. Considering the inevitability of retrograde urinary infection when these high fistulas, especially those connecting the rectum with the urinary tract, remain open (6), early diagnosis and treatment is of primary importance.

ASSOCIATED ANOMALIES

Additional developmental anomalies were found in 24, or 46.1 per cent, of this series of cases. Other writers (1, 6, 10, 11, 13, 18) have found the proportion of associated anomalies to vary from 28.0 to 68.7 per cent. Of the 24 cases at hand, 19 or 79.2 per cent were males, and 18 or 75.0 per cent were patients with type III anal anomalies. This relatively higher incidence of associated anomalies in males with type III anal defects has been noted previously (10). These additional malformations involved the genitourinary system most fre-

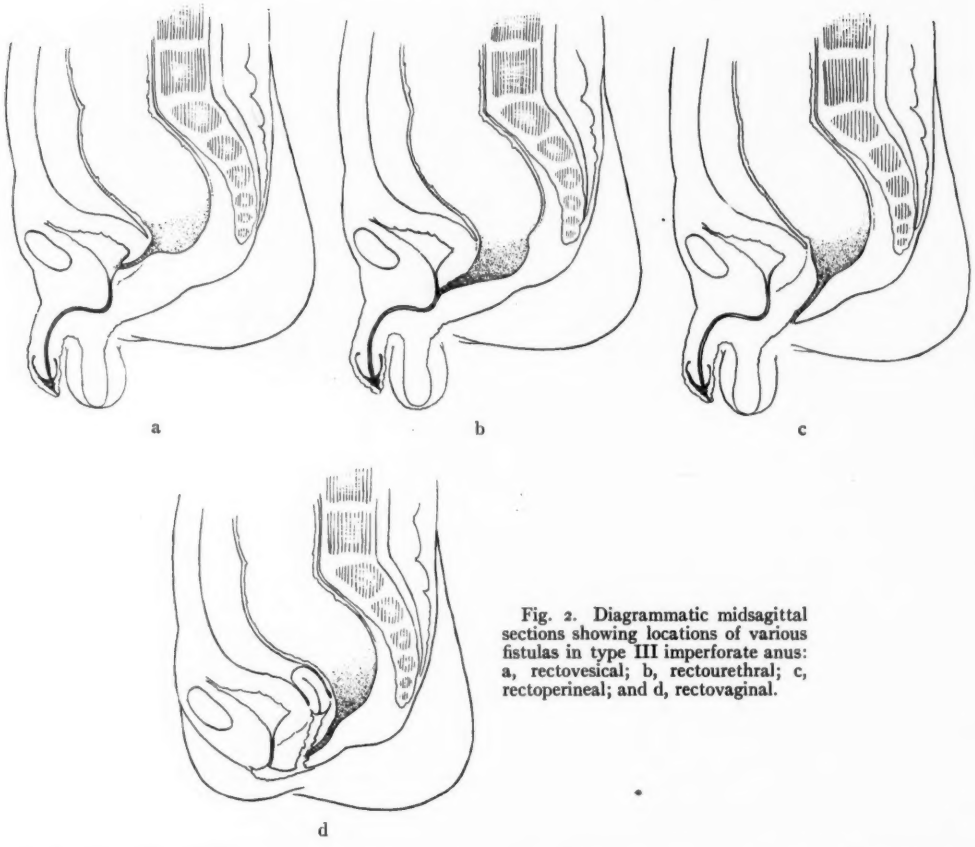


Fig. 2. Diagrammatic midsagittal sections showing locations of various fistulas in type III imperforate anus: a, rectovesical; b, rectourethral; c, rectoperineal; and d, rectovaginal.

quently, occurring in 14 cases. There were 10 additional gastrointestinal malformations, 8 patients with anomalous cardiac development, 4 with skeletal anomalies, 3 with mongolism, and 1 with a defect involving the larynx. The largest number of additional anomalies in any one patient was 5. Death was attributable directly to the associated anomalies in 8 patients; these malformations were contributory in the death of 2. All but 2 of these deaths were in males of type III. This represents a mortality of 15.4 per cent of the entire series. The deaths due to these anomalies are 42.1 per cent of the overall mortality.

Of particular interest are the additional malformations of the gastrointestinal tract, 10 in number, of which 4 were directly responsible for death. Three of the fatal defects were tracheo-esophageal fistulas, the fourth, a duodenal atresia. It is said that anal defects are the developmental lesion most commonly associated with esophageal anomalies. Reports of these concurrent malformations are frequent in the literature (11, 13, 21, 22). Awareness of the association and careful search for a possible esophageal defect is important, especially in males with the type III anal anomaly, since the operative correction of the esophageal lesion must take precedence over that for the anal defect.

TREATMENT OF ANORECTAL MALFORMATIONS

Type I anomaly is treated by repeated anal dilatations, daily at first, decreasing in fre-

quency as the function of the anus approaches normal. Care should be taken to traumatize surrounding tissues as little as possible in order to avoid scar tissue formation. Should the external aperture be very small, cruciate incision of the remaining membrane is indicated. If stenosis is produced by a fold higher in the rectal ampulla, longitudinal division of the structure, followed by transverse closure has been advocated (7). Few patients with this type enter the hospital; only 2 are present in this series. One of these died shortly after birth of congenital heart disease, the other has only fair anal function at the age of 12 years. A firm scar is present at the mucocutaneous junction causing incontinence when the stools become loose.

The treatment of type II malformation has consisted of cruciate incision of the obstructing membrane, or of excision of the membrane and suture of the anal mucosa to the skin. Both procedures are followed by repeated dilatations. Eleven patients were so treated. Two others were admitted for resection of stenosing scars or bands, one following primary treatment elsewhere, the other following spontaneous rupture of the membrane. Eleven of these patients are now living and well with satisfactory anal function, although one required secondary excision of a perianal scar. There were 2 fatalities. One was due to a congenital laryngeal anomaly; the second, who died at home of undetermined cause 1 week following discharge from the hospital, is considered an operative death. Three fistulas were present in this group, all of which were rectoperineal in location, small, and superficial. All closed spontaneously after being opened at the operation and allowed to heal by granulation.

The classic method for treating the majority of cases of type III anal anomaly is a procedure called proctoplasty, or perineoplasty. This operation consists of approaching the rectal pouch from below through a vertical midline perineal incision. The blind rectal end is dissected free from surrounding tissues and drawn downward to be sutured without tension, after opening, to the skin about the anal site. If the primary proctoplasty is unsuccessful, because the operator failed to find the rectal pouch or to mobilize it sufficiently to bring down without tension, high sigmoid colostomy has been advocated. In some cases, usually those in a relatively poor preoperative condition, primary colostomy is performed without an attempt to dissect the perineum.

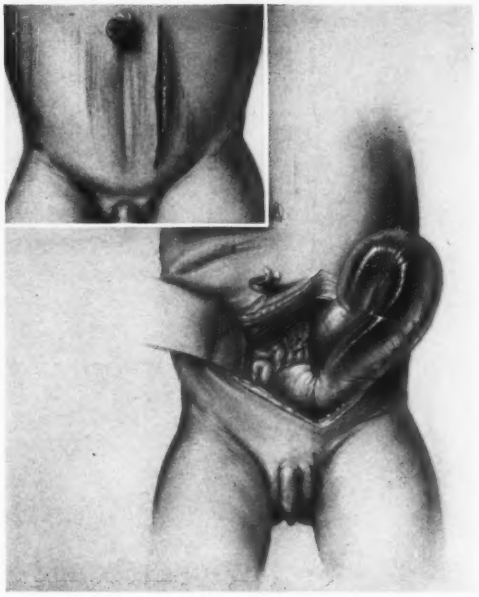


Fig. 3. Technique of abdominoperineal rectal replacement. A left lower rectus, muscle splitting incision is made (inset). The distended sigmoid colon is allowed to eviscerate.

Fistulas occurring with type III imperforate anus are variously treated. Low fistulas (rectoperineal, rectovulvar, and low rectovaginal) have been successfully closed at the time of the primary proctoplasty by division or excision of the fistulous tract and the mobilizing of the rectum sufficiently to exteriorize the rectal end of the fistula (13). Frequently, a low fistula with a stoma adequate for passage of feces is simply dilated to relieve obstruction, correction of the anomaly being postponed until the patient is over 3 years of age. At this time proctoplasty is performed, usually preceded by sigmoid colostomy, which is then closed as a third stage operation at a later date. High fistulas (rectourinary or high rectovaginal) are corrected at an earlier age. Several months after the primary proctoplasty,

Fig. 4. Dissection is carried anteriorly about the rectal pouch and the fistula identified (inset). The tract is then divided between clamps.

a sigmoid colostomy is made, followed after a varying interval by isolation and division of the fistula, usually through an abdominal approach. For rectourinary fistulas, a suprapubic cystotomy is frequently performed with this stage. Later the cystotomy tube is removed and, finally, the colostomy is closed. As the artificial anus tends to contract during the period of disuse, dilatations or plastic repair of the perianal scar are often necessary following closure of the colostomy.

In this series of 37 cases of type III anorectal anomaly, 34 were subjected to primary operations with 7 deaths, a mortality of 20.6 per cent. Table III lists the primary procedures used and the mortality of each.

In the literature one finds that the mortality of primary successful proctoplasty varies from 11.7 to 22.2 per cent (13, 18, 24), that of primary colostomy from 18.0 to 25.0 per cent (18, 24), and that of unsuccessful proctoplasty followed by colostomy from 68.7 to 84.6 per cent (13, 24). Except in this last category, the fatality rate approximates the average range.

In addition to the primary operations of the present series, 11 patients had 40 secondary "stage" procedures performed, with 1 operative death, an additional mortality of 9.1 per

Fig. 5. The rectal pouch is further released by posterior dissection (inset). (The dotted line represents the dissecting finger.) At the completion of this stage of the operation there is sufficient bowel to reach the perineum without tension.

cent. Another patient died of peritonitis secondary to bilateral acute streptococcal salpingitis during the interval prior to closure of her rectovaginal fistula several months after a primary dilatation. Three of these 11 patients are still waiting for further operations, 2 new patients having had the primary procedure await the beginning of the various stages of the fistula repair, while 4 have been lost to follow-up before discharged as cured.

It is evident that, in addition to this primary and secondary mortality, there is a large and incalculable morbidity expressed in readmissions and total hospitalization involved in the classic treatment of type III anorectal anomalies. For this reason we have recently attempted to correct the anal deformity and close the fistula in certain of these cases with one operation, an abdominoperineal replacement of the rectum (Figs. 3, 4, 5, 6, and 7).

The skin of the infant is prepared on all aspects from axillae to below the knees; he is placed upon a sterile sheet and the feet and legs are wrapped separately in sterile drapes. A left lower rectus, muscle splitting incision is made. The dilated loops of colon are allowed to eviscerate and are covered with warm moist packs. Sufficient gas and meconium within the pelvic colon are gently "milked" proximally to allow adequate exposure of the pelvic floor. The peritoneal reflexion is incised about the rectum and the dissection is

Fig. 6. The assistant holds the infant in an exaggerated lithotomy position, and a short vertical, midline incision is made through skin and subcutaneous tissue overlying the anal dimple. A clamp is then placed through the exact center of the dimple, spread gently and the ligated rectal end of the fistula drawn through to the exterior.

carried inferiorly to expose the blind rectal end and the fistula (if one exists). This is divided between clamps and ligated with fairly heavy chromic catgut. The ends of the ligature on the rectal side are left long. Further dissection of the pouch continues until the rectum is sufficiently free to be drawn downward to the perineum without tension. A short vertical incision, through skin and subcutaneous tissue only, is then made in the perineal midline. With a blunt instrument, this opening is connected with the superior area of dissection through the center of the anal dimple, thus saving as much of the anal sphincter as possible. The ligated portion of the rectal pouch is grasped and drawn through the perineal wound. The sigmoid colon completely covers the pelvic peritoneal defect so that no reperitonization is necessary. The abdomen is closed with interrupted figure of eight sutures of fine, stainless steel wire, uniting peritoneum, muscle, and fascial layers. Interrupted sutures of nonabsorbable material are used to close the skin. The ligated end of the rectal pouch is removed with scissors after the full thickness of the rectum has been approximated to the skin of the perineal incision with interrupted nonabsorbable sutures.

TABLE III.—PRIMARY PROCEDURES

Primary operation	Number of infants operated upon	Operative deaths	Operative mortality per cent
Proctoplasty (successful)	11	2	18.2
Proctoplasty (unsuccessful)	3	1	33.3
Colostomy (following unsuccessful proctoplasty)	4	1	25.0
Colostomy (primary)	6	1	16.7
Dilatation of fistula	5	0	0.0
Abdominoperineal replacement	4	1	25.0
Abdominoperineal replacement (following unsuccessful proctoplasty)	1	1	100.0
Totals	34	7	20.6 Average

Preoperative and postoperative care is similar to that practiced in atresias elsewhere in the gastrointestinal tract (23). A Levine tube is passed into the stomach in all cases prior to operation. (A tracheo-esophageal fistula may be discovered at this time.) The stomach is kept empty by means of the tube during the operation and for 12 to 24 hours thereafter. A vein is cannulized prior to operation, and electrolytes, blood, or plasma given as indicated during the procedure. Open drop ether anesthesia is used. Postoperatively, constant intravenous hydration is maintained to give 10 to 15 cubic centimeters of saline per pound per day and total fluids of 50 to 75 cubic centimeters per pound per day. Transfusions of 50 to 75 cubic centimeters are given as indicated. Feedings are started as in the postpyloric regimen after 24 hours and gradually increased as the normal motility of the bowel is attained. Penicillin or streptomycin may be given prophylactically. The anal sutures are removed on the tenth postoperative day. Gentle anal dilatations are begun on the fifth postoperative day and continued daily for several weeks, diminishing in frequency as the anal function approaches normal.

It is felt that the abdominoperineal replacement of the rectum is indicated in all early cases of type III anorectal anomaly with evidence of rectourinary or high rectovaginal fistula, and in all such cases without evidence of fistula, since both the incidence and the error in diagnosis of such fistulas in these patients are known to be high. However, when these

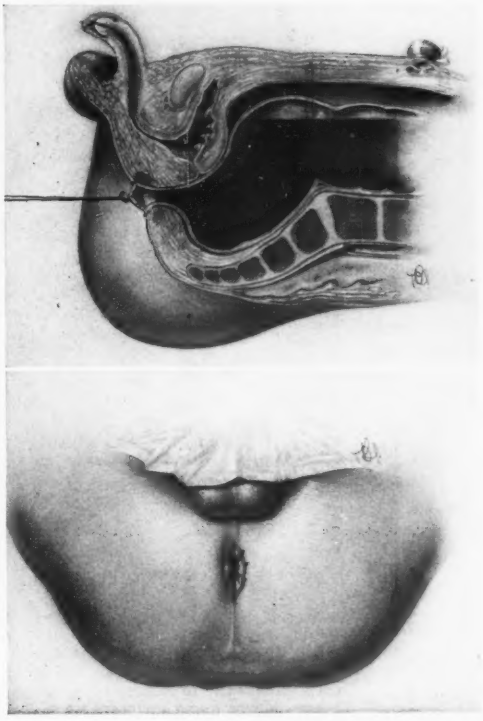


Fig. 7. a, above, The full thickness of the rectum is approximated to the perianal skin and the excess, ligated portion of the bowel removed. b, below, Illustrates the final appearance of the anal region and perineal wound.

infants are seen late (over 60 hours of age), especially if considerable abdominal distention is present, they are probably best treated by primary proctoplasty or colostomy, depending upon the general condition and the distance between the perineum and the rectal pouch as determined by roentgenograms. In such cases, where there is little abdominal distention, the abdomen may be opened as for the abdominoperineal procedure. If, however, sigmoid distention precludes adequate exposure of the pelvic floor, a high sigmoid colostomy may be carried out and the abdomen closed. Cases of type III anomaly with low, narrow, rectovaginal or rectoperineal fistulas should have a primary proctoplasty with division and exteriorization of the fistula. Less preferably, a primary dilatation of a wider

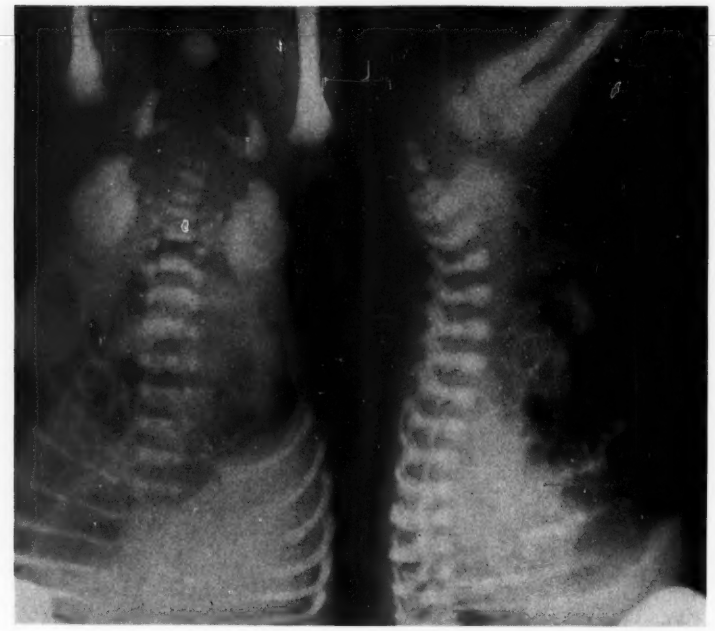


Fig. 8. Roentgenogram of infant with type III imperforate anus at the age of 12 hours, using the Wangensteen and Rice technique. Gas had not entered the pelvic colon sufficiently to localize the rectal pouch. At operation, shortly afterward, the rectosigmoid was found to terminate slightly below the pelvic peritoneal reflexion. A rectourinary fistula was present.

low fistula may be carried out, followed by colostomy and secondary proctoplasty at a later date. The primary abdominoperineal replacement, or some other single stage operation, is probably indicated in type IV anorectal anomalies when seen early, although we have had no experience with them. The operation is definitely contraindicated following attempted, unsuccessful proctoplasty.

This is a preliminary report in that the primary abdominoperineal replacement of the rectum has been performed in only 4 cases. One infant died due to an associated anomaly. The second had normal postoperative bowel and anal function but his course was marked by recurrent periods of cyanosis, fever, and occasional abdominal distention. There was a marked and unexplained anemia. A gastrointestinal series at about 5 weeks of age revealed no anatomic nor functional abnormality, and there was no evidence of peritonitis. After 7 weeks the infant expired. Unfortunately, no autopsy was obtainable. Although we include this as an operative death, an associated cardiac anomaly appears to be the more likely cause. The 2 other patients are living and well at the ages of 4½ years and 10 months, respectively. The anal function of the elder is normal, the perianal scar is soft and a small but functioning sphincter is present. The younger patient has no evidence of an anal sphincter, and no voluntary control has yet appeared. The anus admits the fifth finger with ease and the scar remains moderately soft. The operative mortality is, therefore, 25 per cent up to this time.

ROENTGENOGRAPHY PRIOR TO OPERATION

Because of the nature of the abdominoperineal procedure, the earlier the patient comes to surgery, the less the distention and the simpler the operation. The Wangensteen and

TABLE IV.—RESULTS

Type of anomaly	Total cases of each type	Anal function				Cases between operative stages	Deaths (all causes)	Lost to follow-up
		Normal	Good	Fair	Poor			
I	2			1			1	
II	13	9	2				2	
III	37	3	3	4	2	5	16	4
Totals	52	12	5	5	2	5	19	4

TABLE V.—MORTALITY

Anomaly type	Total mortality	Operative mortality	Mortality from associated anomalies	Mortality from other causes
I	50.0%	0.0%	50.0%	0.0%
II	15.4	7.7	7.7	0.0
III	43.2	23.5*	16.2	5.4

*Figure is based on infants operated upon and includes fatalities following both primary and secondary operations.

Rice technique for obtaining roentgenograms of the abdomen with the infant held head down and a marker at the anal dimple has been found inaccurate before the age of 24 hours by numerous observers (1, 12, 13, 14, 18, 31). The films of 3 of our patients, taken at the ages of 2, 12, and 14 hours, respectively, illustrated this error. In each case, at laparotomy shortly following this diagnostic procedure, the rectal pouch was found to be at the level of, or just below, the pelvic peritoneal reflexion. In no case had the gas approached this region, according to the films (Fig. 8). To postpone the operation until accurate roentgenograms may be obtainable increases the hazard to the patient by increasing the difficulty of exposure at the time of surgery. The x-ray procedure is somewhat traumatic, especially if the infant is held head down for some minutes and the abdomen massaged in an attempt to cause gas to enter the rectal pouch (32). The films give no information regarding the presence or location of high fistulas. For these reasons it is felt that preoperative roentgenograms may frequently be omitted in infants seen at an early age, in which the abdominoperineal operation is indicated.

RESULTS OF TREATMENT

The results of the treatment of imperforate anus are difficult to evaluate accurately prior to the age of 2 or 2½ years when toilet habits have developed. If the child has control of evacuation even when the feces are loose (as in diarrhea or after catharsis), normal anal function is considered to have been attained. If control is present when the feces are of normal consistency, and incontinence occurs only with watery stools, the anal function is considered "good." Function is only "fair" if control is lost with soft stools, and "poor" if total incontinence or alternating incontinence and impaction occur. Patients in these last two categories usually require further surgical treatment. The best results are seen in those children who develop a palpable anal sphincter. The preponderance of good results in this series occurred in patients with type II anal anomaly (Table IV).

Some aspects of the mortality have been discussed. Table V presents the fatality statistics in further detail. The total fatality rate for the entire series is 36.5 per cent, the overall operative mortality being 17.3 per cent, that from associated anomalies 15.4 per cent, and from other causes, 3.8 per cent.

SUMMARY AND CONCLUSIONS

The various types of congenital anal anomalies together with commonly associated fistulas are described. Fifty-two cases taken from the services of Drs. L. Chaffin, J. N. Nichols, K. Smiley and the authors have been studied. In the majority of the patients, the rectum was found to end blindly at a varying distance from the perineum, the type III anorectal anomaly. The incidence of fistulas and of associated malformations of other systems is relatively higher within this group of cases than in the other types. The mortality and functional results of treatment are about the same in this series as in others of the literature.

The treatment of the type III case with a rectourinary or high rectovaginal fistula has caused prolonged hospitalization and additional mortality by the use of multiple "stage" operations. A one stage primary procedure has consequently been adopted for certain of these patients. This operation, the abdominoperineal replacement of the rectum, has the advantage of correcting the anal defect and closing the fistula at the same time. The use-

fulness of roentgenograms in locating the terminal rectal pouch is found to be limited in cases in which the abdominoperineal procedure is indicated, due to the early age at which laparotomy is best performed. The indications for the operation are discussed and the results in 4 cases described.

NOTE. Since February, 1948, 7 additional cases of imperforate anus have had the abdominoperineal replacement operation performed. Six of these were of type III and 1 of type IV. One death occurred due to an associated congenital cardiac anomaly. The 6 other patients are living and well with normal lower bowel function except that the extent of sphincter activity cannot yet be established. With these additional cases, the operative mortality becomes 9.1 per cent.

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TRYPSIN AND THROMBIN INDUCED VENOUS THROMBOSIS AND ITS PREVENTION WITH DICUMAROL, 3,3′ METHYLENEBIS (4 HYDROXYCOUMARIN)

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IT is generally accepted that the hypoprothrombinemia consequent to the administration of dicumarol prevents the formation and propagation of venous thrombi. Such acceptance has arisen from three sources. Bingham, Meyer, and Pohle, and Butt, Allen, and Bollman demonstrated that dicumarol given in adequate doses not only depleted the prothrombin content of the circulating blood but also prolonged the actual coagulation time of the blood. Bollman and Preston, Dale and Jacques, and Richards and Cortell demonstrated by a variety of methods that venous thrombosis could be prevented in animals that were dicumarolized. These experiments were successful, however, only when the prothrombin content was reduced to such levels that hemorrhage was commonly observed and was not infrequently a cause of death. A statistical analysis (1) of a large group of patients undergoing surgery with and without the postoperative administration of dicumarol suggested that dicumarolization appreciably reduced the incidence of postoperative thromboembolic complications.

Empirically there is little reason to doubt the efficacy of dicumarolization in the prophylaxis of venous thrombosis. There is no agreement, however, on the optimum prothrombin levels necessary for either the prophylaxis or therapeusis of venous thrombosis. Subjecting this problem to experimental analysis has been difficult, primarily because the customary methods of producing thrombosis have in few respects simulated the events occurring in the human being during the development of venous thrombi. The injection of sclerosing agents or direct mechanical trauma to the wall of a vein does not resemble disturbances to coagulation and blood flow which result in thrombosis in the human being.

Tagnon demonstrated that the commonly observed prolongation of coagulation after the intravenous administration of either trypsin or thrombin was due not to an increase in autogenous heparin but to depletion of the prothrombin content of the blood. Such depletion was apparently the result of the conversion of large quantities of prothrombin to thrombin during the process of intravascular coagulation and diffuse fibrin deposition along the intima of blood vessels. Tagnon's observations suggested that the injection of small quantities of thrombin or trypsin into isolated segments of veins to initiate coagulation might resemble intravascular clotting more closely than other methods; also that a previously induced hypoprothrombinemia might interfere with the formation of thrombi.

MATERIALS AND METHODS

Healthy, mongrel dogs weighing 5 to 9 kilograms were used. Thrombin was dissolved in 0.9 per cent saline at room temperature so that 0.05 cubic centimeter of the solution contained 20 units. Trypsin, 1/250, was dissolved in saline at room temperature so that 0.0001 milligram was contained in 0.05 cubic centimeter of solution. Blood for clotting and prothrombin determinations was drawn immediately before each experiment. Clotting times were determined at room temperature in saline washed tubes after the withdrawal of blood in saline washed syringes. Prothrombin determinations were performed by Brambel's method before dicumarolization and immedi-

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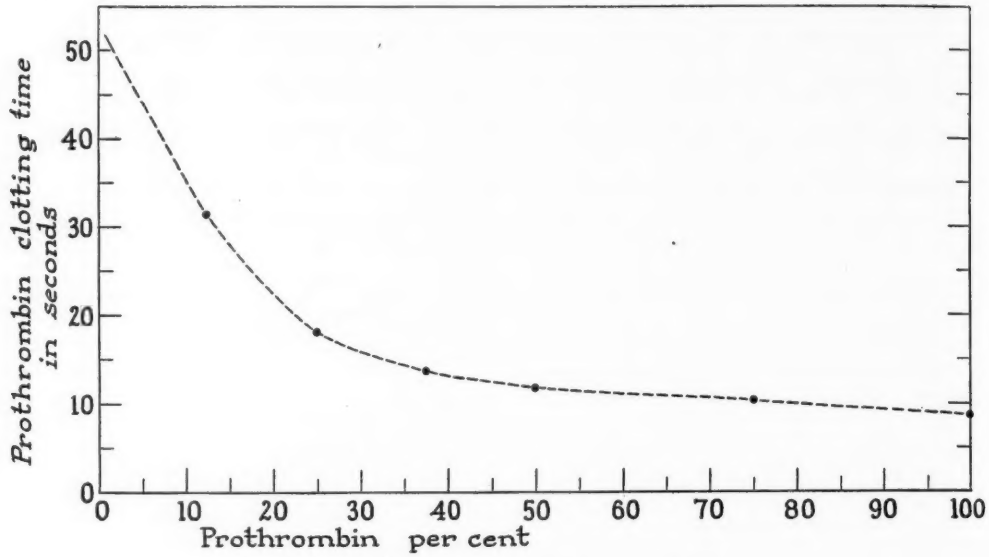

Fig. 1. Calibration curve of plasma from 9 normal dogs.

ately before each experiment. The calibration curve used is seen in Figure 1.

Dogs were lightly anesthetized with intravenous sodium pentobarbital, 0.44 cubic centimeter per kilogram of body weight. The anesthetic agent was never injected into the same vein which was used for carrying out the experiment.

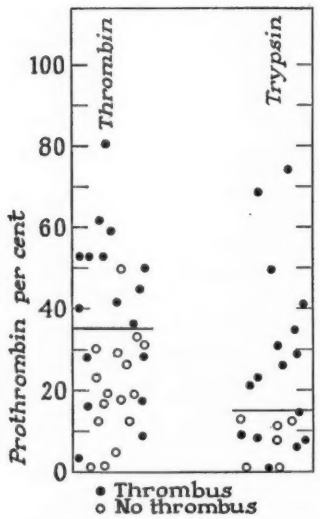

Fig. 2. A graphic representation of thrombosis produced at various levels of hypoprothrombinemia.

Radial veins were used for the thrombin experiments. The external jugular and superficial femoral veins were used for the trypsin experiments. The veins were exposed under aseptic conditions and soft rubber clamps were gently applied to isolate a segment 2.5 centimeters in length. Any branch which occasionally entered the isolated segments was ligated with No. 000 silk. The clamps were applied so that the segment was not fully distended with blood in order that excessive distention after the injection of thrombin of trypsin would not produce intimal disruption. Immediately after the clamps were applied 0.05 cubic centimeter of either the trypsin or thrombin solution was injected through a No. 27 needle into the vein. A gauze sponge was held gently over the site of the needle puncture for 30 seconds. After exactly 15 minutes had elapsed the vein was opened longitudinally. A fresh gauze sponge was placed beneath the vein before the lumen was entered. No thrombus was considered to be present when all the blood flowed

TABLE I.—THROMBIN PRODUCED CLOTS

Experiment No.	Lee-White clotting time in minutes	Prothrombin clotting time in seconds	Prothrombin per cent	Clot present
1.	8.0	13.0	40.0	Yes
2.		11.6	53.0	Yes
3.	13.0	11.6	53.0	Yes
4.	17.0	47.9	2.8	Yes
5.	9.0	17.9	25.2	No
6.	14.0	52.6	0.5	No
7.	9.0	19.6	23.5	No
8.	9.0	23.3	19.5	No
9.	6.0	31.4	12.5	No
10.	6.5	10.9	62.0	Yes
11.	5.5	11.6	53.0	Yes
12.	14.0	25.5	17.0	No
13.	14.0	51.9	1.5	No
14.	14.0	9.8	81.0	Yes
15.	3.5	16.5	29.2	No
16.	10.0	43.9	5.0	No
17.	11.5	11.1	59.0	Yes
18.	7.5	12.4	42.0	Yes
19.	6.5	24.7	17.8	No
20.	7.0	17.4	27.0	No
21.	8.0	14.1	35.5	Yes
22.	9.5	22.5	19.0	No
23.	9.0	16.6	28.0	Yes
24.		11.8	50.5	No
25.	3.0	14.9	33.0	No
26.	11.5	31.4	12.5	No
27.	6.0	25.0	17.5	Yes
28.	2.5	17.9	26.8	No
29.	3.0	17.3	28.0	Yes
30.	7.0	12.4	45.0	Yes
31.	6.5	11.7	51.0	Yes
32.	10.0	39.0	8.0	Yes
33.	6.0	25.5	17.0	Yes

from the vein and was immediately absorbed by the sponge. In some instances a small pin point clot was observed at the site of the vein puncture, but the remaining blood was completely liquid. Any clot other than this was considered to be a thrombus and is so designated in the tabulated results (Tables I and II). In many instances the character of the clot varied from the controls and this fact will be discussed in more detail.

TABLE II.—TRYPSIN PRODUCED CLOTS

Experiment No.	Prothrombin clotting time in seconds	Prothrombin per cent	Clot present
1.	30.5	13.0	No
2.	29.0	14.0	No
3.	20.9	21.9	Yes
4.	20.2	22.9	Yes
5.	39.8	7.6	Yes
6.	51.6	0.5	Yes
7.	51.6	0.5	No
8.	32.0	12.0	No
9.	11.9	50.0	Yes
10.	10.7	69.8	Yes
11.	17.4	27.0	Yes
12.	15.6	31.5	Yes
13.	31.4	12.5	No
14.	10.2	75.0	Yes
15.	14.2	35.0	Yes
16.	16.6	29.0	Yes
17.	29.2	14.0	Yes
18.	41.0	7.0	Yes
19.	39.1	8.0	Yes
20.	36.5	9.2	Yes
21.	39.0	8.0	No
22.	12.8	42.0	Yes

RESULTS

Isolation of a segment of vein with soft rubber clamps in 8 instances did not produce thrombosis in 15 minutes. Isolation plus the injection of 0.05 cubic centimeter of 0.9 per cent saline in 7 instances produced no thrombosis. Fifty-four veins were used to determine the amount of thrombin necessary to produce coagulation. In 10 radial veins that received 20 units of thrombin, clots formed in all. In 14 veins receiving 10 units of thrombin, clots were present in 6, or 43 per cent. Forty-seven jugular and femoral veins were used to determine the minimal amount of trypsin necessary to produce thrombosis; less than 0.0001 milligram produced no thrombi in 6 of 7 instances.

Graphic representations of the results of the injections of thrombin and trypsin in the presence of hypoprothrombinemia may be seen in Figure 2. In the thrombin experiments, thrombi were present in 6 veins and were absent in 15 veins when the prothrombin level

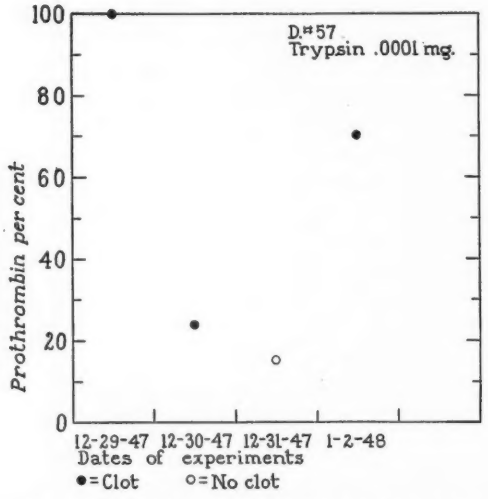

Fig. 3. Thrombosis attempted during the fall and rise of the blood prothrombin level in 1 animal.

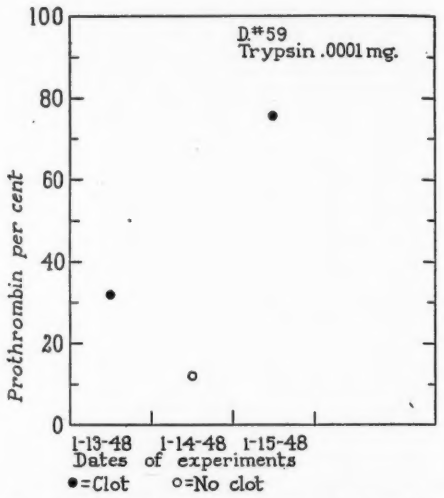

Fig. 4. Thrombosis attempted during the fall and rise of the blood prothrombin level in 1 animal.

was below 35 per cent of normal. When the level was above 35 per cent, thrombi were present 11 times and absent once. A more marked reduction in the prothrombin reserve was necessary to inhibit coagulation when trypsin was used. When the prothrombin per cent was below 15 per cent of normal, thrombi were present 6 times and absent on 6 occasions. Above 15 per cent, however, in 10 instances thrombi were always present. Two individual experiments are shown in Figures 3 and 4.

COMMENT

The results of the experiments indicate that venous thrombosis produced by a combination of venous stasis and the injection of either thrombin or trypsin may be prevented by reducing the blood prothrombin level. Levels below 35 per cent were necessary in the thrombin induced coagulation and levels below 15 per cent were required to inhibit the trypsin induced thrombosis. It cannot be stated that thrombosis could be inhibited routinely below these levels. On the other hand, it can be stated that above these levels, no success was obtained in preventing the formation of thrombi. When coagulation did occur below 35 per cent in the thrombin and 15 per cent in the trypsin experiments, the character of the thrombus was different from the controls. Instead of a firm compact thrombus, a loosely adherent mass of fibrin and red cells resembling sludge was encountered.

In clinical practice we have attempted during the past 2 years to maintain the prothrombin level at 20 per cent of normal in the prophylaxis and treatment of venous thrombosis.

CONCLUSIONS

1. This study suggests that venous thrombi experimentally produced may be prevented by reducing the blood prothrombin level below 35 per cent of normal with dicumarol.

2. Blood prothrombin levels above 35 per cent were ineffectual in preventing the formation of thrombi.

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EVALUATION OF HEXACHLOROPHENE AND DETERGENTS AS SUBSTITUTES FOR THE SURGICAL SCRUB

A Biological Technique

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THE present study was undertaken in order to evaluate some of the newer cleansing and bactericidal agents as possible substitutes for the conventional surgical scrub.[1] A technique which would eliminate much of the time and irritation that are undesirable features of the 10 minute scrub would obviously be worth while. The agents selected for evaluation included a bactericidal agent[2] incorporated in soap, and two detergents[3, 4].

The literature contains several articles on hexachlorophene (1, 7, 8, 9), all giving favorable reports on the use of this agent incorporated in soap for cleansing skin. The techniques utilized by these workers were essentially variations of Price's method for obtaining colony counts from hands and forearms.

In this study animal inoculation was used to evaluate the agents tested. The examination of hand washings by animal inoculation tends to (a) differentiate potentially pathogenic from purely saprophytic organisms, and (b) to eliminate false bactericidal effects of chemicals only bacteriostatic in nature (3, 4).

To establish a maximum standard reaction of animals to pathogenic bacterial infection the method of Nungester, Jourdonais, and Wolf (5) of introducing mucin to decrease the resistance of animals to given infection was utilized. Repeated titrations in which a virulent culture of hemolytic streptococci was injected intraperitoneally into mice, with and without mucin, revealed that the animals' resistance was reduced about one million fold when mucin was used. Whereas, no less than 0.5 milliliter of the culture was lethal; 0.5 milliliter of a 10^{-6} dilution of the same culture, when combined with mucin, provided a fatal dose. Strict saprophytes do not kill mice when injected with mucin.

Four series in all were undertaken. The first two were of a preliminary nature and compared a routine surgical scrub, with the use of bar soap, with a two minute wash with the use of either duponal, phisoderm, or hexachlorophene, no brush being used with the last three agents. One of these series was carried out in the hospital, the subjects being physicians actively engaged in clinical work on the surgical wards and in the operating rooms. The series was short and did not contribute any significant comparative results. However, it did reveal the remarkably low pathogenicity of hand rinsings obtained prior to cleansing. The mean mortality of mice injected with hand rinsings obtained prior to cleansing was 18 per cent as against a mean of 13 per cent mortality produced by all the samples taken after cleansing. The results were derived from 12 cleansings with each agent, all agents being tested concurrently. The differences between the improvements resulting from the use of each cleansing agent were not significant.

In the second series, physicians engaged in laboratory duties contaminated their hands with 5 milliliters of a hemolytic streptococcal culture before cleansing with the same agents used in the first series as noted previously. Contamination of the hands and wrists was done in order to provide a greater killing power to the rinsings injected and to place a more severe test upon the cleansing and bactericidal properties of the agents tested. As has been noted, the pathogenicity of bacteria usually present on one's hands and forearms

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[1]Control cleansings, as well as surgical scrub, employed bar soap (ivory).

[2]Hexachlorophene (G-11), Hexachloro-, di-hydroxy-, diphenyl methane, 2 per cent incorporated in bar soap of the lux type.

[3]Phisoderm (2) (regular type) containing sodium t-octylphenoxy ethoxyethyl ether sulfonate.

[4]Duponal (WA paste) 15 per cent in water.

is remarkably low and to simulate the unusual, contamination was undertaken.

In this series, each agent, duponal, phisoderm, hexachlorophene, and soap and brush, was used for 15 cleansings. The data for all agents were obtained concurrently. The results obtained in this second preliminary series again suggested that favorable cleansing could be obtained with either detergent or with hexachlorophene soap as compared with the surgical scrub. The mean mortality of animals injected with rinsings taken after contamination and before cleansing was 79 per cent, as compared with a mean mortality following cleansing of 27 per cent for all agents tested. Here again the variations between the agents tested were not significant.

The efficacy of this biological method was well illustrated by data derived from the second preliminary series, in which contamination of the hands was done prior to cleansing. Twenty-four random autopsies were done on animals that had died after having been injected with samples obtained following cleansing. Cultures from the spleens of these animals revealed hemolytic streptococci in 10 cases, whereas, in vitro cultures of the peptone water samples that had been injected into these same 10 animals failed to reveal hemolytic streptococci in 5 instances. There is little doubt that in many instances the abundant overgrowth of bacteria accompanying postmortem changes precluded the identification of hemolytic streptococci. This does not devaluate the animal inoculation method as all mice dead following injection provide the critical numbers for statistical analysis whether streptococci are or are not identified in the animals.

As a result of these preliminary experiments, two more series were conducted comparing the routine surgical scrub with a 3 minute brushless wash with soap containing hexachlorophene. In the second of these two series the hands and forearms were contaminated prior to cleansing.

PROCEDURE

1. Each subject cleansed with a given agent (10 minute surgical scrub or 3 minute wash with hexachlorophene soap) on each of 5 successive days. The 10 minute surgical scrub was routine. The 3 minute wash with bar soap containing 2 per cent hexachlorophene involved lathering from fingertips to elbows for 1 minute, cleaning fingernails with an orangewood stick for 1 minute while the lather remained on the arms; and a third minute was spent relathering the arms and hands, which were then rinsed in running tap water.

2. Each subject rinsed his hands in 200 milliliters of sterile 0.5 per cent peptone water for 1 minute, 20 seconds being spent rubbing the palms together, 20 seconds rubbing the back of one hand, and 20 seconds rubbing the back of the other hand.

3. Immediately thereafter, 5 milliliters was taken from each basin of peptone water rinsings and mixed with 5 milliliters of sterile 5 per cent hog gastric mucin. One milliliter of this mixture was then injected intraperitoneally into each of 10 mice. The animals were observed for 5 days and deaths recorded.

In the series in which contamination was done, 5 milliliters of a 24 hour blood-brain-heart broth culture were placed in the palms of each subject who then rubbed the culture over all the skin of the hands and wrists. The culture air-dried for 3 minutes after being applied. Such contamination was done prior to cleansing.

COMPARISON OF THE SURGICAL SCRUB WITH BRUSHLESS CLEANSING WHEN SOAP CONTAINING 2 PER CENT HEXACHLOROPHENE IS USED

A statistically significant difference in their cleansing effect on normal skin. Twelve subjects were used, all with "scrubbing" experience, 11 with at least a year's surgical training. After an individual used each agent, at least 10 days intervened before his use of the other agent. The subjects used neither antibacterial agents nor prolonged (i.e., 10 minutes) scrubbing of their hands during this interval. This was to allow the skin flora to be re-established (6) and to remove all hexachlorophene from the skin. Seastone has detected traces of hexachlorophene on the skin 48 hours after exposure to this agent in amounts sufficient to act as a bacteriostatic agent, the agent being carried from the hands through the wash water to the culture medium.

TABLE I.—INTRAPERITONEAL INJECTION, WITH MUCIN, OF PEPTONE RINSINGS TAKEN AFTER CLEANSING

Type of cleansing	Hexachlorophene	Routine surgical scrub
No. cleansings	36	42
No. mice injected	348	445
No. dead	28	56
Mortality, per cent	8.0	12.5

P—less than 4%

Table I presents the data from this series. Results show 12.6 per cent mortality following the use of the 10 minute surgical scrub and 8.0 per cent mortality after a 3 minute wash with soap containing 2 per cent hexachlorophene. This difference is statistically significant.

Their value in decreasing transient streptococci. In this series contamination was done as follows:

The skin was contaminated on Monday, 5 milliliters of the 10 milliliters in each hemolytic streptococcal culture being placed on the hands of a subject using one agent and the other 5 milliliters being placed on the hands of the subject using the other. On the 4 subsequent days, no contamination of hands was done. The rest of the method was precisely as in the previous series. Six subjects participated in this experiment, each using both agents, with at least 10 days intervening between their use.

These data are presented in Table II. It should be noted that the culture used in these tests was more virulent than the strain of hemolytic streptococcus originally employed. The noteworthy variance is that present in the death rate of the animals injected with rinsings obtained on the first and second days subsequent to the day of contamination. This difference is apparently due to the persistence of the artificially implanted transient bacteria after 3 days of scrubbing, whereas there was a rapid depression of the pathogenicity of the rinsings from those subjects employing hexachlorophene bar soap. The difference in mortality on the day after contamination is statistically highly significant. In addition, a similarly significant difference persists on the second day after contamination.

TABLE II.—INTRAPERITONEAL INJECTION, WITH MUCIN, OF PEPTONE RINSINGS TAKEN AFTER CLEANSING, CONTAMINATION WITH 5 C.C. OF STREPTOCOCCUS HEMOLYTICUS CULTURE ON FIRST DAY OF SERIES

	Hexachlorophene 3 minute wash (6 subjects)			Routine surgical scrub 10 minutes (7 subjects)		
	No. of mice injected	Dead	Per cent	No. of mice injected	Dead	Per cent
First day	60	59	98.5	70	69	97
Second day	60	7	11.7	70	27	38.6
Third day	60	9	15	70	32	45.7
Fourth day	60	5	10	70	9	15
Fifth day	60	8	16	70	13	21.7

The possibilities of using hexachlorophene soap for preparing the operative field are apparent. Not only would mechanical cleansing be obtained, but the added bactericidal effect would be advantageous. In order to compare the bactericidal effect of hexachlorophene on skin as compared with tincture of iodine, an animal method was used. This was a modification of that used by Kempf and Nungester (4). These authors contaminated a mouse's tail with either a virulent culture of pneumococci or streptococci and, after applying the antiseptic agent to be tested, inserted the amputated tip of the tail into the animal's peritoneal cavity. Our method used the abdominal skin as the test area.

METHOD

1. Under ether anesthesia the abdomen was shaved and swabbed with a 24 hour blood-brain-heart broth culture of hemolytic streptococci.

2. After a 1 minute interval as measured by a sand glass, the agent being tested (either tincture of iodine, U.S.P. or a 1 per cent solution of the monopotassium salt of hexachlorophene in 33 per cent ethyl alcohol) was blotted on the contaminated area. Care was taken not to wipe away the culture when applying the iodine or hexachlorophene solution.

3. After a measured 2 minute interval a 1 by 0.5 centimeter elliptical segment of skin was removed and the peritoneum opened. The skin segment was placed in peritoneal cavity.

TABLE III.—RESULTS

	No. animals	Deaths	Mortality per cent
Iodine	36	15	42
Hexachlorophene	36	24	67
Control	16	15	94

4. Five-tenths milliliter of sterile mucin was injected intraperitoneally and the wound then closed with cotton sutures.

These animals were observed for 5 days and deaths recorded. A control series was conducted concurrently in which the method was identical except for the application of iodine or hexachlorophene. Table III presents the results of this experiment.

The 42 per cent mortality is similar to that obtained by Kempf and Nungester in their tests in which the animals' tails were used to carry the culture and agent into the peritoneal cavity. Their mortality was 38 per cent when iodine was used and from 85 to 95 per cent when merthiolate, mecresin, or phemerol was used. These agents were not compared directly in our experiment.

DISCUSSION

These experiments substantiate our recommendation that the routine surgical scrub be eliminated in favor of a 3 minute wash with soap containing hexachlorophene. Such a change is desirable because of the common irritating effects resulting from repeated cleansing with a brush. The low pathogenicity of the skin flora from the hands and forearms is noteworthy. This is indicated by an 18 per cent mortality of mice injected with mucin and skin washings obtained prior to cleansing.

It should be noted that no alcohol rinse was employed in these tests. Attention is drawn to Seastone's statement that the use of an alcohol rinse after cleansing may extract part of the hexachlorophene from the skin.

In conducting this work, an attempt was made to obtain subjective criticism of the different agents being evaluated. Several subjects found duponal somewhat drying and irritating to the skin although no definite sensitivity was noted objectively. Phisoderm was well received, as was the bar soap containing hexachlorophene. No instances of sensitivity to either were observed.

Although data are not sufficient to draw fixed conclusions on the basis of preliminary experiments, cleansing with phisoderm (2) and duponal would also appear to have possibilities as substitutes for the surgical scrub. A combination of a detergent and hexachlorophene might be efficacious. In addition, use of these agents for preparing the operative field is suggested.

SUMMARY

1. A biologic method for evaluation of agents for cleansing the skin is presented. This technique differentiates the bacteriostatic and bactericidal action of chemicals. The use of animals whose species resistance has been greatly lowered by injection of mucin also serves to determine the potential virulence of skin flora.

2. Under the conditions of these experiments it may be concluded that the use of bar soap containing 2 per cent hexachlorophene for 3 minutes without a brush is superior to the routine two brush, 10 minute surgical scrub.

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TENDON TRANSPLANTS FOR IRREPARABLE RADIAL NERVE PARALYSIS

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THE present paper is based on the personal experience of the author as Chief of Orthopedic Surgery and later as Chief of the Surgical Service while at McGuire General Hospital, Richmond, Virginia.

In all 45 cases of tendon transplantation for irreparable radial nerve paralysis were carried out by the author from February 27, 1945 to March 9, 1946. Various operative procedures were used, and a clinical evaluation of the procedure made at the time that the patient was being either separated from the Service or transferred to another installation.

REVIEW OF LITERATURE

Operative attempts for the restitution of function by tendon transplants in radial nerve paralysis date back over 50 years.

In 1896, Drobnik did a transplant for the paralysis of the posterior branch of the radial nerve with conservation of the function of the extensor carpi radialis longus. The extensor carpi radialis longus was placed into the common extensors of the fingers.

In 1897, Rochet had a case of partial paralysis of the radial nerve with persistence of function of the extensor carpi radialis longus which was transplanted into the extensor pollicis longus and extensor indicis proprius.

In 1902, Cappelli did a shortening of the extensor carpi radialis longus and transplantation of the flexor carpi ulnaris into the common extensors of the fingers, and of the palmaris longus into the extensor indicis proprius.

From the end of the nineteenth century to World War I, sporadic case reports and questionable procedures appeared in the literature. In 1917, Sir Robert Jones standardized his operation of tendon transplants for radial nerve paralysis and firmly established a milestone in the method of treatment of this condition. He transplanted the pronator teres into the extensor carpi radialis longus, the flexor carpi radialis into the extensor pollicis longus and the extensor indicis proprius, and the flexor carpi ulnaris into the extensor tendons of the third, fourth, and fifth fingers.

In civilian life injuries which are of the type to produce irreparable radial nerve paralysis are few and far between. Consequently, in the intervening period between World War I and World War II, the only report of a number of cases seen by any one surgeon or any one clinic was from the Mayo Clinic where 23 cases were treated over a period of 26 years. This article written by Young and Lowe gave an excellent review of a long term follow-up of patients.

Bonola, in 1936, presented an excellent review of the subject and also an ingenious method of evaluating these cases from a functional standpoint.

During World War II, due to the kind of warfare with previously unknown types of high velocity shells, land mines, and aerial bombs, the injuries to the extremities reached astronomical figures. Consequently, in a relatively short period of time, a substantial number of injuries of this type gravitated to specialized centers.

In 1946, Altman and Trott reported on 28 cases from McCloskey General Hospital, in this country, and Zachary reported on 57 cases from Wingfield, Morris Hospital in England. These cases were done by various surgeons on the staff.

Lamphier and Littler, in 1947, reported on an undisclosed number of the cases performed at Cushing General Hospital. The procedure they used was the transference of the flexor carpi radialis into the abductor pollicis longus, extensor pollicis longus and brevis, and of the flexor carpi ulnaris into the common extensor tendons to the fingers.

In cases reported by Altman and Trott the pronator teres was inserted into the extensor carpi radialis longus and brevis, the flexor

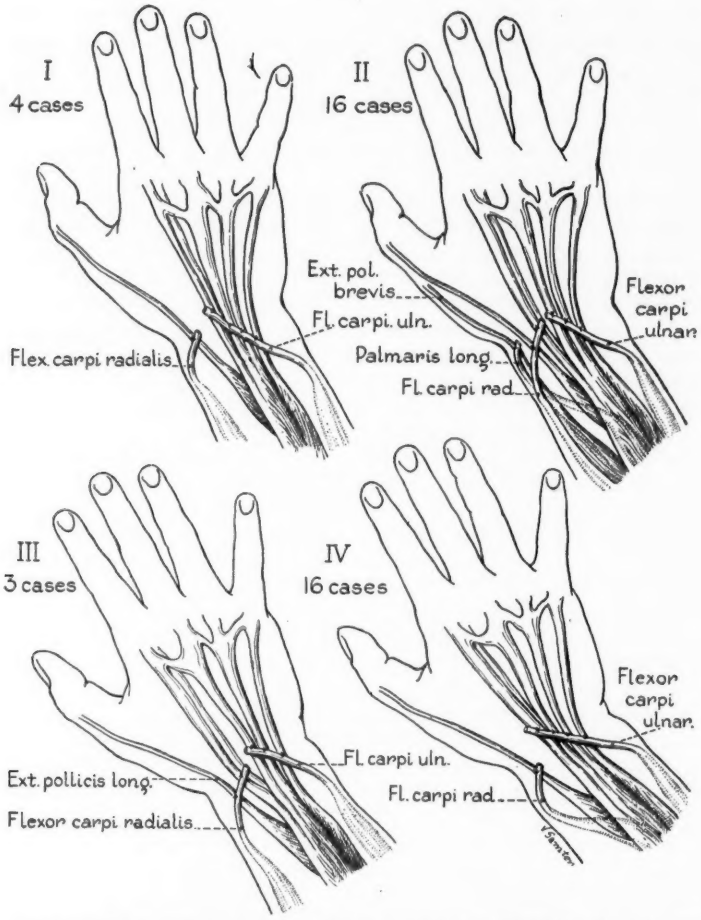

Fig. 1a. The most common types of tendon transplants performed in this series are graphically shown in illustrations I, II, III, and IV. The number of each type of operation performed is listed under the Roman numerals.

carpi radialis into the extensor pollicis longus and brevis, and the flexor carpi ulnaris into all the extensor tendons of the fingers. They reported uniformly good results in their cases.

In most of the cases reported by Zachary, the pronator teres was transplanted into the extensor carpi radialis longus, the flexor carpi ulnaris into the extensor tendons of the third, fourth, and fifth fingers, and the flexor carpi radialis into the extensor pollicis longus and extensor indicis proprius. In a careful analysis of these cases, he brought out a very important point, with which the writer was able to concur. He noted that if all the flexors of the wrist, flexor carpi ulnaris, flexor carpi radialis, and palmaris longus, were transferred, stabilization of the wrist was lost in many instances and often led to incomplete extension of fingers and unnecessary weakening or abolishment of voluntary power of wrist flexion.

REPORT OF PERSONAL CASES

Various methods of tendon transplants were used in this series and careful after studies

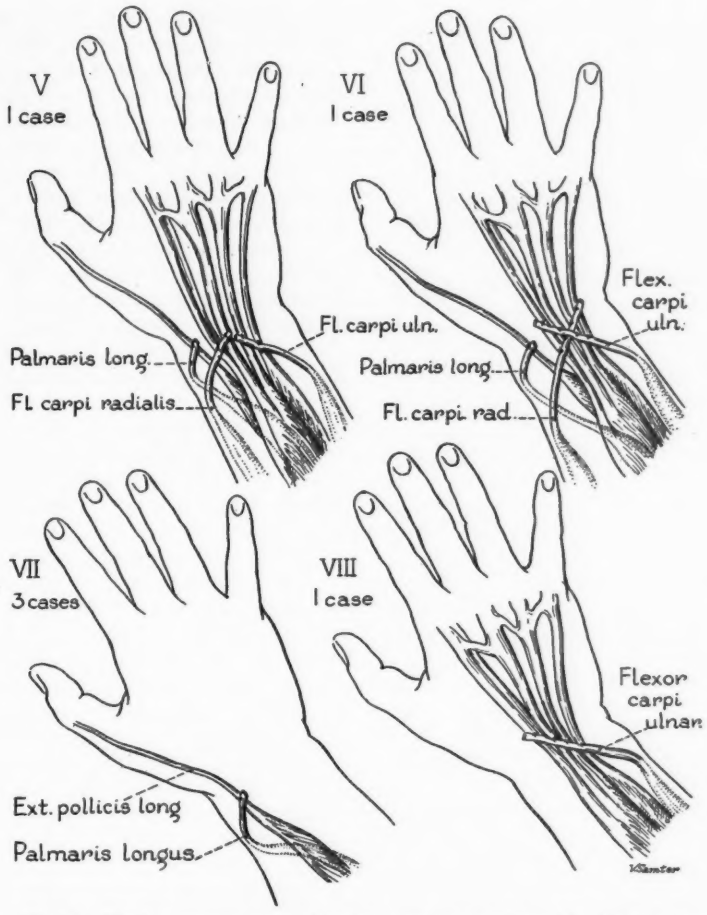

Fig. 1b. The less common types of tendon transplants performed in this series are graphically shown in illustrations V, VI, VII, and VIII. This group consists of 6 cases.

were made in order to evaluate their relative efficiency.

Forty-five cases were carried out in all. The various types of transplants are presented in Table I and in Figures 1a and 1b.

The study of these patients during the convalescent periods and the analysis of many of the cases by means of movies revealed the following:

1. Combined suture of one transplanted tendon into two or more inactive tendons having different functions (such as suturing the palmaris longus or the flexor carpi radialis into the extensor pollicis longus and brevis, or extensor pollicis longus and extensor indicis proprius) was not as satisfactory as transference into a single thumb tendon.

2. In spite of experimental evidence to the contrary, the flexor carpi ulnaris muscle is perfectly capable of extending the wrist and all the fingers, if properly sutured under the correct amount of tension. (Sudeck, in 1944, stated that the working power of the flexor carpi ulnaris was 2 kgm. and that this was

TABLE I

	Transplanted tendons	Recipient tendons	No. of cases
I.	Flexor carpi radialis Flexor carpi ulnaris	Extensor pollicis longus Common extensor tendons of fingers	4
II.	Flexor carpi radialis Flexor carpi ulnaris Palmaris longus	Extensor pollicis longus Extensor indicis proprius Extensor tendons of the third, fourth and fifth fingers Extensor pollicis brevis	16
III.	Flexor carpi radialis Flexor carpi ulnaris	Extensor pollicis longus Extensor indicis proprius Extensor tendons of third, fourth and fifth fingers	3
IV.	Flexor carpi ulnaris Palmaris longus	Extensor tendons of second, third, fourth, and fifth fingers Extensor pollicis longus	16
V.	Flexor carpi radialis Flexor carpi ulnaris Palmaris longus	Extensor tendons of second, third fingers Extensor tendons of fourth and fifth fingers Extensor pollicis longus	1
VI.	Flexor carpi radialis Flexor carpi ulnaris 2 operations performed—unsuccessful case Palmaris longus	Extensor tendons of second, third, fourth, and fifth fingers Extensor tendons of second, third, fourth and fifth fingers Extensor pollicis longus	1
VII.	Palmaris longus (Paralysis of extensor and abductor tendons of the thumb)	Extensor pollicis longus	3
VIII.	Flexor carpi ulnaris (Thumb muscles were normal—partial radial nerve paralysis)	Extensor tendons of second, third, fourth and fifth fingers	1
		Total cases	45

sufficient only for extending the digits, as 4 to 5 kgm. of power were required to extend the wrist as well as the digits.)

3. It is most important that the transplanted tendons run in as straight a line as possible from the origin of the muscle to the insertion of the recipient tendon. Acute angles or long sweeping curves of the transplanted tendons decrease their efficiency.

4. The complete severance of the extensor pollicis longus tendon in the "anatomical snuff box" and the swinging of the loose end anteriorly, then suturing it to the transferred palmaris longus tendon, gives a most excellent combined extensor and abductor motion to the thumb. In fact, it is almost impossible to distinguish the result from the normal thumb function. This observation was accidentally discovered in 1 case as a result of severance of the palmaris longus tendon a little too proximal to its insertion, so that it was relatively short and could not reach the extensor pollicis longus tendon in its normal location.

5. Although the transplantation of the flexor carpi radialis into the extensor pollicis longus and extensor indicis proprius, and the flexor carpi ulnaris to the extensor tendons of the third, fourth, and fifth fingers gives a wide opening of the thumb and fingers in the extended position, it weakens and in some cases abolishes voluntary flexion of the wrist if the palmaris longus tendon is absent. When the palmaris longus tendon is present (absent in 3 of the 45 cases) it motorizes the thumb very well if sutured to the severed extensor pollicis longus tendon as described previously.

6. All of the tendon transferences done in this series produced marked improvement of the function of the hand regardless of what minor detail of tendon to tendon transference was performed. Only 1 case was considered as a complete failure. Whether or not it was a surgical or psychosomatic failure will probably never be definitely known. This was the case of an officer who did not desire to be separated from the Service until the September semester of college began the following year. He was operated upon July 19, 1945. It was impossible to get his co-operation in physiotherapy and occupational therapy postoperatively. A second operation was performed on January 31, 1946 for a second tendon transference. By that time such marked fibrosis had resulted in the fingers that the case was ultimately classed as a failure.

PREOPERATIVE CARE

Unless the wrist, fingers, and thumb have free joint motion, the surgeon is handicapped before he starts. It is most important that the hand receive intensive physical therapy in the form of whirlpool baths, heat, and massage until all the joints are freely movable, and the skin soft and pliable. If adhesions of the joints exist prior to surgery, it will be almost impossible to correct the joint limitation after surgery. None of the patients in this series were operated upon until the fist could be firmly made actively, and full extension of the fingers and thumb was possible, passively.

OPERATIVE TECHNIQUE

The day prior to surgery the finger nails are cut short, the hand and arm shaved up to the

elbow, and the extremity washed thoroughly with soap and water. After 10 minutes of good washing with frequent changes of water, alcohol and ether are poured over the part and sterile towels are then placed around the arm from the finger tips to the elbow.

The day of surgery, when the patient is on the operating table, the extremity is again washed with soap and water, then drenched with alcohol and ether. A sterile towel is now placed over the field and the arm elevated. All of the blood is squeezed out of the arm by the use of a Martin constrictor to the upper arm where a blood pressure cuff is pumped up to 300 millimeters of mercury to insure hemostasis. The Martin constrictor is then removed. This leaves a bloodless extremity to be worked upon, gives excellent visibility, is a great time saver, and avoids the unnecessary trauma to exposed tendons and their sheaths from repeated sponging.

The field is now prepared with one of the standard operating room antiseptics.

The insertions of the palmaris longus, flexor carpi radialis, and flexor carpi ulnaris are easily identified by extension of the wrist. Short longitudinal incisions are made, the tendon isolated and then cut free. By pulling gently on the cut tendon, its proximal portion can be palpated in the forearm, 6 to 8 inches proximally, and then through individual short longitudinal incisions, each tendon is exposed. The tendon is then drawn from the distal end proximally so that it is freed through the proximal incision. The flexor carpi ulnaris has a very low attachment of muscle fibers frequently down to the last one-half inch of the tendon. The muscle should be sufficiently removed so that about 3 inches of the tendon is free of muscle in order to permit better suture into the recipient tendons and also to provide for better gliding between the subcutaneous fat on one side and the fascia of the arm on the other side.

The hand and forearm are then pronated and the extensor pollicis longus tendon isolated in the "anatomical snuff box." It is completely severed at the level of the distal end of the radius. It is most important that this tendon be accurately identified before it is severed. Retraction of the proper tendon

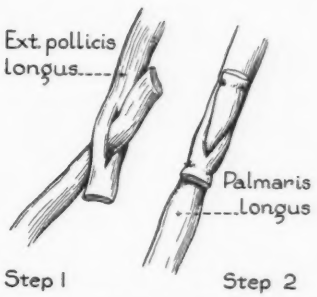

Fig. 2. The buttonhole technique for the suture of the palmaris longus to the extensor pollicis longus was most satisfactory in the author's experience. Step 1, a slit is made in the extensor pollicis longus just distal to the cut end, and the palmaris longus tendon is drawn through the slit. Step 2, four silk sutures are then used to hold the tendons together. This type of anastomosis is simple, effective, and rapidly performed.

produces extension of the terminal phalanx of the thumb.

A well known principle of tendon transplantation must be kept in mind at this stage of the procedure. A transplanted tendon and muscle will work most efficiently if its action is in a straight line running from the origin of the motor muscle to the insertion of the recipient tendon. Any sharp curves or angulations decrease or completely defeat the transplanted motorized unit. For this reason the author has always attempted to attach the transplanted tendons as far distally as possible. This, by necessity, required the exposure of the common extensor tendons at the level of the dorsal carpal ligament. This overlying portion of the ligament was routinely removed, so that the anastomosis of the tendons was given the smooth gliding anterior portion of the ligament on the anterior aspect and the abundant subcutaneous fat on the posterior aspect. On no occasion were any adhesions detected in this area, nor was there ever seen any posterior or mediolateral displacement of the extensor communis tendons. A number of men have recommended that the tendon anastomosis always be done proximal to the dorsal carpal ligament. This procedure angulates the transplanted tendon and thereby decreases its mechanical efficiency. All of the transplanted tendons are tunneled between the fascia of the forearm on one side and the

subcutaneous fat on the more exterior side. This gives an excellent gliding mechanism.

In 20 cases of this series the flexor carpi ulnaris was sutured to all of the common extensor tendons. It was found to be an efficient motorized unit provided that a few points were carefully adhered to.

The flexor carpi ulnaris tendon works best if the anastomosis to the common extensor tendons of the fingers is done so the tendon to the little finger is sutured approximately ¾ inch proximal to the suture of the tendon to the index finger. In this fashion the same amount of extension tension could be placed on all of the extensor tendons. The ring and middle finger tendons are anastomosed between the index and little finger tendons so that they maintain the same angle of suturing (the middle finger tendon being sutured ¼ inch distal to the level where the ring finger was being sutured Fig. 2).

The amount of tension necessary for adequate function is very difficult to determine. Experience is the best teacher. If too much tension is used the wrist cannot be adequately flexed. If not enough tension is used dorsiflexion of the wrist is inadequate.

In none of the cases treated in this series was any ulnar deviation of the wrist noted when extension was initiated by the flexor carpi ulnaris muscle.

When the flexor carpi radialis is used, it is drawn subcutaneously around the radius and inserted into the desired thumb or index tendon. The same principles apply to this tendon as to the flexor carpi ulnaris.

After it was once discovered that if the extensor pollicis longus tendon was severed at the "anatomical snuff box" and drawn anteriorly and anastomosed to the palmaris longus giving an excellent combined thumb function of extension and abduction, the procedure alone was used for the rehabilitation of the thumb. In 3 cases of this series the palmaris longus was absent, and then the flexor carpi radialis was used.

At the completion of this anastomosis, the line of action must be straight from the origin of the palmaris longus muscle to the insertion of the extensor pollicis longus tendon into the terminal phalanx of the thumb.

It was the author's experience that combined anastomosis into the extensor pollicis longus, or brevis, and abductor pollicis longus, or any combination of anastomosis of these tendons to a single motorized transplanted unit, decreased the efficiency of the unit considerably. Combined pulls of two or more tendons on the thumb do not work as efficiently because each tendon has a slightly different line of pull, and besides has a different amplitude of movement to produce a desired motion of a digit. This point has been well brought out by Bunnell.

Various methods of anastomosis of the tendons were tried. The anastomosis giving the maximum holding power with the fewest number of stitches is by necessity the one desired. The final type of standardized anastomosis used in this series is as follows:

In anastomosing the palmaris longus tendon into the severed extensor pollicis longus tendon, the buttonhole technique is used. A small slit is made through the extensor pollicis longus tendon near its severed end, and the palmaris longus tendon is drawn through this opening. The tendons are then sutured to one another by two sutures at the end of each tendon making a side-to-side anastomosis (Fig. 2).

When the anastomosis is made for the flexor carpi ulnaris or radialis, the recipient tendons are buttonholed at the desired level for the anastomosis and a straight Kelly forceps inserted through all the buttonholes. The motorized tendon is then slit longitudinally and half of it is drawn through the recipient tendons with the forceps. Two interrupted silk sutures are used to fix each tendon. The other half of the slit motorized tendon is then laid over its other half and sutured in place with only two silk sutures at the proximal and distal end of the anastomosis. This gives an excellent mechanical anastomosis with a minimal number of stitches. On one occasion it was necessary to reopen this type of anastomosis in order to put more tension on the motorized tendon. The inspection of the anastomosis showed no excessive scar tissue production, and the anastomosis was covered with what appeared to be a normal tendon sheath (Fig. 3).

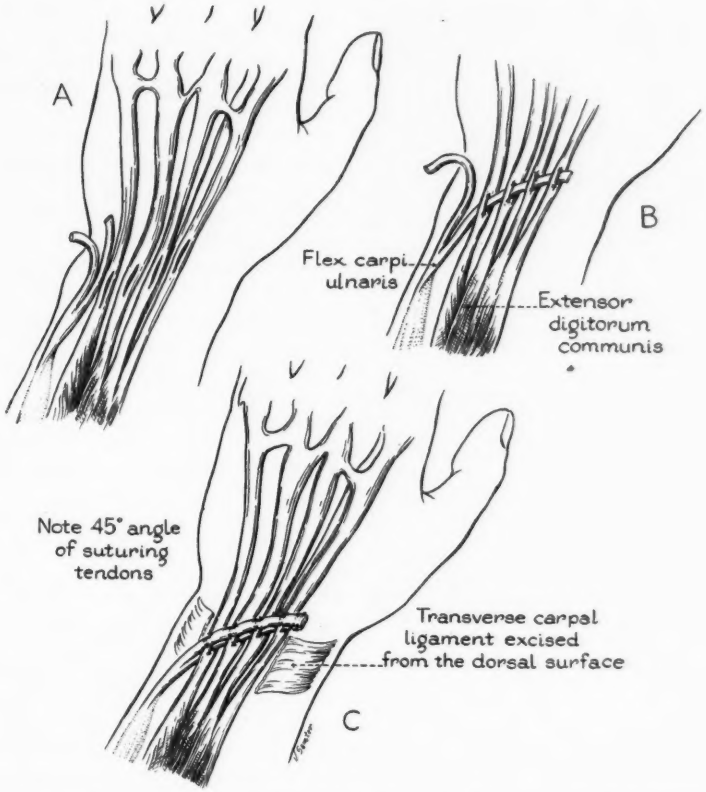

Fig. 3. A, Shows the buttonholing of each extensor tendon at an angle of 45 degrees so that the tendon to the little finger has the most proximal buttonhole, and the extensor tendon to the index finger has the most distal slit. B, The flexor carpi ulnaris tendon is slit longitudinally, and half of the tendon is threaded through the buttonhole slits and sutured in place. C, The transverse carpal ligament is completely excised, and the remaining half of the flexor carpi ulnaris tendon is sutured over the top of the tendons. This gives excellent security of the suture line.

POSTOPERATIVE CARE

The postoperative care of these patients is most important. Immobilization in plaster for 16 to 18 days postoperatively is necessary in order to insure good healing of the tendons and also to prevent excessive formation of scar tissue. Earlier immobilization of the tendons produces more adhesions and scar tissue. This was shown experimentally by Mason and Allen.

A cast is used extending from the proximal interphalangeal joint to the elbow with the wrist in the cock-up position and the thumb in the mid-abduction extension position.

At the end of 16 to 18 days physical therapy is begun. This consists of muscle re-education, whirlpool baths, heat, and gentle massage. The importance of daily physical therapy by a competent therapist cannot be over emphasized. The individual gains confidence easily, muscle re-education becomes simplified, normal interphalangeal and metacarpophalangeal motion is maintained, and a shorter convalescence results.

At the end of 4 weeks from the date of operation, occupational therapy should be instituted. Graduated types of work, from the sim-

Fig. 4, above. Preoperative film of a drop wrist following an irreparable radial nerve paralysis. The fingers cannot be voluntarily extended, nor can the thumb be abducted or extended.

Fig. 5, center. The postoperative motions of the thumb 3 months after the palmaris longus tendon was sutured to the severed extensor pollicis longus tendon. Full abduction-extension motion is now possible.

Fig. 6, below. The postoperative motion of the fingers in the same case. The flexor carpi ulnaris was sutured to all the common extensor tendons of the fingers. Note the marked hypertrophy of the palmaris longus tendon running to the thumb.

ple to the complicated, and from the light to the heavy, should be carried out. By the aid of a good occupational therapist the muscular hypertrophy and the co-ordination of the wrist, fingers, and thumb improves. Usually if the patient is co-operative, a relatively good hand results in about 3 to 4 months from the date of surgery (Figs. 4, 5, 6). In most cases improvement reaches its peak within a period of approximately 9 months. In a few cases which were performed prior to World War II in civilian practice and which were observed for a few years thereafter, the improvement was very little, if any, after the initial 9 months' period.

CONCLUSIONS

1. The transplantation of the palmaris longus tendon into the severed extensor pollicis longus tendon gives an excellent result of abduction-extension power to the thumb. It is most important that the pull of the palmaris longus be in a straight line from the origin of the palmaris longus to the insertion of the extensor pollicis longus tendon into the terminal phalanx of the thumb.

2. The transference of the flexor carpi ulnaris into the extensor tendons of the second, third, fourth, and fifth fingers gives good finger and wrist extension. The flexor carpi ulnaris must be sutured at a 45 degree angle to the long axis of the extensor tendons, so that the anastomosis to the extensor tendon of the second finger is about ¾ to 1 inch distal to the anastomosis to the extensor tendon of the fifth finger.

The use of the flexor carpi radialis into one of the thumb tendons and the extensor tendon of the index finger is not advocated unless there is an absence of the palmaris longus tendon. Its transference in addition to the flexor carpi ulnaris and the palmaris longus tendons weakens or abolishes voluntary wrist flexion, weakens the stabilization of the wrist so that complete extension of the fingers is difficult, prolongs the operative procedure, and adds an unnecessary additional operative risk.

3. The transplanted tendon must exert its pull in as straight a line as possible from the origin of the muscle to the insertion of the recipient tendon. Sharp angles or curves decrease the efficiency of the motorized unit.

4. The transplantation of a single tendon into the extensor pollicis longus is far more efficient than transplantation of a single tendon into two or more thumb, or thumb and index finger tendons. Variation of amplitude of each tendon to perform its normal function and the difference of the line of pull correspondingly decrease the efficiency of the unit.

5. Preoperative physical therapy must be carried out until complete range of motion is possible in the interphalangeal and metacarpophalangeal joints of the thumb and fingers. The flexion motion can be performed actively while the extension motions of the fingers and the abduction-extension movements of the

thumb can, of course, be performed only passively.

Transplanted tendons are unable to overcome the restricted movements which are the result of periarticular adhesions of the joints. Time spent in preoperative physical therapy pays the greatest dividends and enhances the chances for success in this procedure.

6. Postoperative physical therapy and later occupational therapy of increased gradation of amplitude and stress shorten the convalescence. Muscle hypertrophy is quicker, coordination of fingers and thumb motions are learned in less time, and the morale of the patient is kept at its highest level.

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RUPTURED UTERUS

A Report of 42 Cases

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DURING the 20 years from 1928 to 1947 inclusive, there were 42 instances of ruptured uteri at the Cook County Hospital. In this same period 92,226 women were delivered of viable infants. Although rupture of the uterus is rare, it carries an exceedingly high mortality for both mother and baby and therefore the subject seems worthy of review.

In the present series there were 23 maternal deaths, or a maternal mortality of 54.76 per cent. The fetal mortality was 79.07 per cent—28 stillborn infants, 1 neonatal death, and 5 infants who died in utero undelivered. Nine infants lived, a fetal saving of only 20.93 per cent.

In 16 cases the uterus ruptured before the women were brought to the hospital and in 26 instances the rupture occurred in the Cook County Hospital. Twenty-two of the mothers were white and 20 were negro.

Table I shows the distribution as to ages. It will be noted that the largest number of cases was in the age group of 36 to 40 years, inclusive, and the next largest in the 21 to 25 year age group.

Thirty-eight ruptures were in multiparous and 4 ruptures were in nulliparous women (Table II).

The individual circumstances vary so much as to etiology, symptoms, degree of shock, diagnosis, and treatment, that it is almost impossible to make graphs of any value. We are, therefore, stating briefly the salient facts in each case. An effort has been made to group the abstracts as to etiology.

In 9 cases rupture occurred following previous cesarean section. In the first 7 reported, rupture occurred after previous classical sections and in Cases 8 and 9 after low cervical sections. (In patients 6 and 8 the original sections were performed at the Cook County Hospital.)

CASE 1 (1933). White secundipara, tertigravida, aged 21 years, at term, cephalic presentation. Bag of waters was intact. The patient had been in labor for 11 hours, progressing normally to 7 centimeters and station plus 2, when she suddenly grabbed her abdomen and cried out, "I'm tearing." Profound shock ensued. Supravaginal hysterectomy was immediately performed and a transfusion of 1,000 cubic centimeters of blood given. She died of peritonitis on the 11th day after operation. The infant was stillborn.

CASE 2 (1933). White secundipara, tertigravida, aged 32 years, 38 weeks' gestation, cephalic presentation. The patient was admitted in mild shock, not in labor. Bag of waters was intact. She had sudden severe constant abdominal pain with marked tenderness over scar and easily palpable fetal parts. Blood transfusion was followed by prompt hysterectomy. Recovery was uneventful. She returned home on the 16th day after operation. The infant was stillborn.

CASE 3 (1940). White primipara, secundigravida, aged 19 years, 38 weeks' gestation, cephalic presen-

TABLE I.—AGE

No. cases	Years
3	Under 20
10	21–25
5	26–30
7	31–35
16	36–40
1	Over 40

TABLE II.

No. of cases	Para
4	0
6	1
3	2
8	3
4	4
6	5
4	6
0	7
3	8
2	9
2	10

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tation. The patient was admitted in early labor and mild shock. Bag of waters was intact. She had marked tenderness over the old scar, and fetal parts were easily palpable. Supracervical hysterectomy was performed, and a transfusion of 1,000 cubic centimeters of blood was given in the operating room. The patient recovered after a stormy postoperative course. The infant was stillborn at 8½ months.

CASE 4 (1941). Colored primipara, secundigravida, aged 21 years, 38 weeks' gestation, cephalic presentation. The patient was admitted in severe shock, not in labor. Bag of waters was intact. During routine visit to prenatal clinic, she had suddenly developed acute pain over an old incision and was hospitalized at once. The infant was removed from the uterine cavity and hysterectomy performed. The patient recovered. The infant lived.

CASE 5 (1946). Colored primipara, tertigravida, aged 32 years, at term, cephalic presentation. The patient entered the hospital in good condition in mild labor for 3 hours. Bag of waters was intact. There were no symptoms of rupture but tear was discovered during laparotomy. Porro section was performed for rupture in upper uterine segment. Penicillin and blood were given postoperatively. The patient recovered and was discharged from the hospital on the 12th day after operation. The infant lived.

CASE 6 (1944). Colored tertipara, quadrigravida, aged 30 years, 30 weeks' gestation. The patient was admitted in severe shock with cramps and pains of 7½ hours' duration. She had believed herself to have "indigestion" for which she had taken castor oil. Sudden sharp abdominal pain followed (Fig. 1). Immediate hysterectomy was performed, and blood and plasma transfusions were given. The patient recovered and returned home on the 17th day after operation. The infant was stillborn.

CASE 7 (1928). White primipara, secundigravida, aged 21 years, 34 weeks' gestation. The patient was admitted in good condition in early labor with a history of previous classical section for generally contracted pelvis. Bag of waters intact. Dilatation was 2 centimeters, station minus 4. Preparations were completed for a repeat section but the patient refused surgery. The uterus ruptured 8½ hours after admission. Laparotomy was carried out immediately after rupture with the patient in poor condition. The patient died during surgery. Baby and placenta were in the peritoneal cavity; there was a 15 centimeter separation of the fundus with smooth edges. The infant was stillborn.

CASE 8 (1934). Colored primipara, secundigravida, aged 16 years, at term, breech presentation. The patient was admitted in mild labor with fetal heart tones present until bag of waters ruptured. Labor pains ceased and were replaced by a steady, not severe, pain which increased in severity in the lower abdomen near the old scar. As soon as the pain became severe laparotomy was done and hysterectomy performed. The patient recovered and returned home on the 12th day after operation. The infant was stillborn.

Fig. 1. Case 6. Tear at site of old section scar. Note smooth edges.

CASE 9 (1942). Colored quadripara, quintigravida, aged 22 years, at term, cephalic presentation. The patient was admitted in mild labor; bag of waters was intact; fetal heart tones were present. There were no symptoms of rupture. During repeat cesarean section, a 2½ centimeter tear in the lower uterine segment was found with the bag of waters bulging through. The section was completed, the uterus resutured and the patient sterilized. The patient recovered. The infant lived.

Of these patients who had undergone previous cesarean section, 6 were in labor and 3 were not upon admission. Two of the patients had such mild symptoms that the rupture of the old scar was not suspected (Cases 5 and 9) but was discovered at the operating table at the time of repeat section.

Six uteri ruptured with transverse presentation.

CASE 10 (1930). White quadripara, quintigravida, aged 38 years. The patient was admitted with the bag of waters ruptured and hand and cord prolapsed. The duration of labor was unknown; fetal heart tones were absent. Delivery was accomplished by decapitation, the fetal trunk was removed, and the head

extracted with a tenaculum without difficulty. Vaginal examination revealed laceration of the right broad ligament. There was no bleeding and no packing was inserted. The patient was not operated upon. She died 3 days postpartum of serofibrinous peritonitis. The infant was stillborn.

CASE 11 (1932). White quintipara, sextigravida, aged 31 years. The patient was admitted with the bag of waters ruptured and hand prolapsed. The duration of labor was unknown, but a private physician had attempted version and extraction in the home. The cervix was completely dilated on admission and fetal heart tones were absent. Decapitation was done, placenta was not expelled, and exploration revealed that it was not in the uterus. Immediate laparotomy was performed for complete laceration extending from the cervix to within 2 centimeters of the fundus. The patient died of peritonitis on the 7th day after operation. The infant was stillborn.

CASE 12 (1933). White quintipara, septigravida, aged 24 years. The patient was admitted with the bag of waters ruptured and hand prolapsed, fetal heart tones were absent. She had been in labor for 7 hours. Delivery was effected by version and extraction, following which she immediately went into shock. Immediate hysterectomy was carried out. She recovered and returned home on the 15th day after operation. The infant lived.

CASE 13 (1940). White quintipara, sextigravida, aged 30 years. The patient was admitted after having been in labor for 10 hours 15 minutes. Fetal heart tones were absent, bag of waters was ruptured. There was slight vaginal bleeding, the head was high on the right, the detached placenta was felt on the left, and a tear was felt in the uterine wall. The abdominal aorta was compressed by the examining hand until hysterectomy was performed. The patient made an uneventful recovery and returned home on the 13th day after operation. The infant was stillborn.

CASE 14 (1943). White secundipara, tertigravida, aged 33 years, transverse presentation. The patient was admitted after having been in labor for 3 hours. The bag of waters was intact. She had had a vaginal plastic and Sturmdorf operation 4 years before. The patient was in the operating room being anesthetized for cesarean section when the cord and arm prolapsed. Version and extraction were carried out through the completely dilated cervix. Immediate total hysterectomy was performed, the tear in the posterior vaginal wall was sutured; 1,500 cubic centimeters of blood and 500 cubic centimeters of plasma were administered. The patient died 20 hours after operation. The infant died.

CASE 15 (1928). White tertipara, quadrigravida, aged 36 years. The patient was admitted with a history of marked respiratory distress and of cyanosis for 2 days. The bag of waters was ruptured; fetal heart tones were questionable. She was in labor for 24 hours with severe pains. When the pains ceased sudden "cardiac decompensation" followed. Morphine sulfate, atropine, and digitalis were administered. Twelve hours after admission the patient was losing ground. The cervix was 8 centimeters dilated, the hand prolapsed, a version and extraction was done without difficulty. The patient died immediately following delivery; there was a tear in the right fundus. The stillborn infant weighed 10 pounds 2 ounces.

Two of the women with transverse presentations and known dead babies were delivered by decapitation, 1 was delivered by cesarean section, and 3 by version and extraction. All were in labor when they arrived at the hospital. There were 4 stillbirths, 1 neonatal death, and 4 mothers were lost. In Case 11 version had been attempted by a physician in the home; this patient died of peritonitis on the seventh postoperative day. In Case 14 a previous vaginal plastic operation had been performed and a cesarean section was planned. While the patient was being anesthetized for surgery, a cord and arm prolapsed. A vaginal examination revealed complete dilatation, and a version and extraction procedure was done, resulting in a tear in the lower uterine segment. In spite of immediate surgery and the administration of 1,500 cubic centimeters of blood and 500 cubic centimeters of plasma, the patient died of shock postoperatively. In Case 15, apparently rupture had occurred at home and the subsequent shock was erroneously interpreted as cardiac decompensation. Had her true condition been recognized on admission to the hospital, she might have been saved with blood transfusions and prompt surgery.

There were 3 other cases of version and extraction, Cases 16, 17, and 18, which, with Cases 12, 13, and 14, total 6 cases of ruptured uteri attributable to version and extraction.

CASE 16 (1929). White quintipara, sextigravida, aged 37 years, cephalic presentation, station minus 3. The patient was admitted after having been in labor at home for 20 hours and attended by a physician. The bag of waters had ruptured 16 hours previously. She had had no pains for 1 hour before admission. Fetal heart tones were absent; there was moderate bleeding from the placenta previa. The cervix was dilated manually, version and extraction were carried out with forceps on the aftercoming head. The patient went into shock. Sterile vaginal examination revealed a tear. Surgery was not done. The patient recovered and returned home on the 16th postpartum day. The stillborn infant weighed 8 pounds 8 ounces.

CASE 17 (1933). White decipara, duodecigravida, aged 33 years, cephalic presentation. The patient was admitted with the cord prolapsed and having been in labor for 7 hours. Fetal heart tones were absent. Delivery was accomplished 8 hours later by version and extraction when dilatation was complete. Rupture of the lower uterine segment was found. Immediate hysterectomy was performed followed by blood transfusion. The patient died of peritonitis on the 10th day after operation. The infant was stillborn.

CASE 18 (1928). White septipara, octigravida, aged 38 years. The patient was admitted in hard labor, pains 2 to 3 minutes apart. Fetal heart tones were present but disappeared during labor. Twelve hours later the cervix was completely dilated, station minus 1. Attempted delivery with Kielland forceps failed; version and extraction followed. Exploration revealed a tear in the right lower uterine segment; immediate hysterectomy was performed. The mother died of bronchopneumonia 2½ days after operation. The infant was stillborn.

Ironically, Case 16 was mishandled according to good obstetric practice, but the mother survived. In the presence of bleeding from what was thought to be a placenta previa, the cervix was *manually dilated* and version and extraction were performed. This procedure is to be condemned. In Case 17, the cervix was completely dilated when the version was performed, but the patient died of peritonitis on the tenth postoperative day. Craniotomy was the procedure of choice in this latter instance since the baby was known to be dead with the cord prolapsed for 8 hours. In Case 18, a craniotomy was also preferable to version, since the baby was known to be dead and forceps delivery had failed. There is no excuse for jeopardizing the life of the mother when it is known that the baby is dead.

There were 4 cases in which oxytocics seem to have been the cause of rupture; 1 patient was given ergot and 3 patients were given pituitrin, all before admission to the Cook County Hospital.

CASE 19 (1929). White nonipara, decigravida, aged 38 years, cephalic presentation, floating. The patient was admitted after having been in labor at home for 4 days. She had been attended by a midwife who gave her "black medicine." On admission the patient had no pains; fetal heart tones were absent. Sterile vaginal examination revealed a cervical tear extending beyond the reach of the examining hand. Immediate hysterectomy was performed. The patient died of hemorrhagic purulent peritonitis on the 5th day after operation. The infant was stillborn.

CASE 20 (1938). White sextipara, nonigravida, aged 36 years, face presentation. The patient was admitted in shock after having been in labor at home for 36 hours. Fetal heart tones were absent. On the afternoon of admission her physician had called and given her ½ cubic centimeter of pituitrin, following which she had had violent pains which stopped suddenly. Delivery was effected by version and extraction followed by blood transfusion. The patient died 2 hours and 50 minutes after admission before surgery could be performed. The infant weighing 11 pounds 7 ounces was stillborn.

CASE 21 (1939). Colored tertipara (one set of twins), tertigravida, aged 34 years, The patient was admitted with head and shoulders delivered after labor of 3½ hours at home. Her physician had administered 1 ampul of pituitrin and was unable to deliver the other shoulder. En route to the hospital the other shoulder delivered spontaneously. The patient went into shock 25 minutes after delivery was completed in the hospital. Two blood transfusions were given for shock but she did not respond. She died 2 hours and 15 minutes following rupture of lower uterine segment. The infant weighing 12 pounds 6¼ ounces was stillborn.

CASE 22 (1940). White sextipara, septigravida, aged 38 years. The patient was admitted in severe shock after having been in labor at home for 27 hours. Her physician stated that dilatation had been complete for 8 hours; he had given her pituitrin 2 minums twice. Four hours after the second dose the patient had had sharp pain causing her to double up. Laparotomy was carried out immediately and blood transfusions were given for shock. She survived this surgery and on the 28th day after operation returned home with a vesicovaginal fistula. She died of ascending infection 6 months later following a genitourinary operation when ureteral transplants were made into the sigmoid. The infant was stillborn.

In the ruptures attributed to oxytocics, all 4 mothers and 4 babies lost their lives. In Case 20, the management of the patient after she arrived in the hospital in shock is to be criticized. The history of violent pains following the administration of ½ cubic centimeter of pituitrin and then of sudden cessation of the pains makes the diagnosis of ruptured uterus obvious. A version and extraction procedure was contraindicated. Treatment of the shock and immediate laparotomy was the procedure of choice. What small chance the woman had to survive was destroyed by the additional trauma of the version and the failure to operate upon her promptly.

There are 6 cases which can be classified as more or less prolonged labors and, probably, disproportions.

Fig. 2. Case 27. Gauze marks oblique tear.

CASE 23 (1932). White octipara, nonigravida, aged 38 years, frank breech presentation. The patient was admitted in shock after having been in labor for 20 hours. Bag of waters had ruptured 1 hour before onset of labor. She had had severe pains for 6 hours before admission. The uterine contractions stopped suddenly. Embryotomy was carried out; the leg amputated with a Braun hook; forceps to aftercoming head. Hysterectomy was performed for a tear in the lower uterine segment. The patient recovered after a stormy postoperative course and returned home on the 36th day after operation. The infant weighing 10 pounds was stillborn.

CASE 24 (1933). Colored quintipara, sextigravida, aged 32 years. This patient had been in labor for 29 hours while in the hospital with a ruptured bag of waters when she suddenly went into shock. The fetal head went out of the pelvis and fetal parts were easily palpable. Supravaginal hysterectomy was performed for the oblique tear in the lower uterine segment. She recovered and returned home on the 21st day after operation. The infant weighing 7 pounds 7 ounces was stillborn.

CASE 25 (1933). White quadripara, quintigravida, aged 36 years. The patient was in hard labor for 20 hours; the cervix was dilated to 9 centimeters; suddenly the pains stopped; the presenting part left the pelvis and the patient went into shock. Immediate laparotomy was performed for the tear in the cervix and left broad ligament. Her postoperative course was stormy and she returned home on the 21st day after operation. The infant was stillborn.

CASE 26 (1934). Colored decipara, undecigravida, aged 35 years. Upon admission after having been in labor for 16 hours, her pulse was 150, bag of waters had ruptured, the cervix was completely dilated, head floating. Shock progressed as the patient was being set up for delivery, the fetal parts were then easily palpable and the fetal heart tones disappeared. Laparotomy was performed immediately for the tear through the right lower uterine segment. Placenta and baby were in the peritoneal cavity. The patient died of peritonitis on the 6th day after operation. The infant weighing 9 pounds was stillborn.

CASE 27 (1942). Colored nullipara, primigravida, aged 22 years. The patient was in labor for 62 hours 30 minutes during which time the fetal heart tones disappeared. Delivery was effected by craniotomy and cleidotomy. She was given supportive postpartum treatment but rupture was not diagnosed (Fig. 2). She died of sepsis on the 21st day postpartum. Postmortem examination revealed a tear of the anterior fundal wall and pelvic peritonitis. The infant was stillborn.

CASE 28 (1944). Colored nullipara, primigravida, aged 20 years. Upon admission patient had hypertension; she had been in mild labor for 15 hours with head floating. Low cervical section was carried out for disproportion (conjugata vera was 7.5 cm., biparietal 10 cm.) and a tear was found in the lower uterine segment. The uterus was resutured. The patient recovered and returned home on the 12th day after operation. The infant lived.

All but 1 of the babies in the group of prolonged labors were stillborn, and 2 mothers were lost. Four of these patients were multiparous (para 3, 5, 8, and 10), illustrating a point that is well known but often forgotten, namely, that a multiparous patient will not necessarily deliver successfully simply because she has previously delivered. In 2 of these, Cases 23 and 26, rupture probably occurred outside of the hospital, but 3 were overlooked in our own labor rooms. In Case 24, a quintipara was allowed to labor for 29 hours without proper evaluation.

There were 2 primiparas among the patients who had prolonged labors; one, Case 27, was in labor 62 hours, delivered by craniotomy and the mother died of peritonitis on her twenty-first postpartum day; the other primipara, Case 28, was delivered by cesarean section and a tear was discovered in the lower uterine segment in the course of the surgery. The uterus was resutured, a questionable procedure, and the patient made an uneventful recovery.

There were 2 disproportions due to hydrocephalus in multiparous patients, one a secundipara and one a sextipara, resulting in the deaths of both mothers and both babies.

CASE 29 (1930). Colored sextipara, septigravida, aged 33 years. This patient was admitted in mild labor with pains 10 minutes apart. Twelve hours later, the uterus became tetanic and the classical signs of threatened rupture were present. A craniotomy was performed for the hydrocephalus; 12 yards of gauze packing was used for the postpartum hemorrhage. The patient died on the delivery table of hemorrhage from a cervical tear extending into the broad ligament. The infant was stillborn.

CASE 30 (1944). White secundipara, tertigravida, aged 34 years, cephalic presentation, floating. The patient was admitted with severe pains after 40 hours of labor at home. The fetal head was perforated through the 4 centimeter cervix with a Smellie perforator; 500 cubic centimeters of serous fluid escaped and the head collapsed. Shock ensued 30 minutes after the head was perforated. The patient was given oxygen, plasma, aminophylline. She did not respond to shock treatment and died undelivered 1 hour 25 minutes after admission. The tear in the lower uterine segment was discovered at postmortem examination.

In 2 cases there appeared to be a defect in the fundus of the uterus.

CASE 31 (1934). Colored nullipara, primigravida, aged 38 years. The patient was admitted not in labor, with bag of waters intact, fetal heart tones not heard. The patient had had 1 day periods for 5 months followed by amenorrhea for 3 months; progressive abdominal enlargement and persistent abdominal cramps. Her condition had been diagnosed as fibroids or ovarian cyst. At laparotomy the fetus was protruding through the the fundus, with the top of the fundus gone. Recovery was uneventful and she returned home on the 10th day after operation. The infant lived.

CASE 32 (1932). Colored tertipara, quadrigravida, aged 21 years. The patient was admitted in shock, not in labor, at 5 months' gestation. Her pregnancy had been normal except for occasional pain until 1 hour before admission when she had had intense abdominal pain, vomited, and fainted. Her abdomen was tympanitic, distended and peristalsis was absent; the cervix was soft, closed and there was a mass up to the umbilicus. Immediate supracervical hysterectomy was performed for perforation of the uterine wall by the placenta. The uterine wall was paper thin. Recovery was uneventful and the patient returned home on the 11th day after operation. The infant was stillborn.

In the 38 year old nullipara, the symptoms and findings were misleading and the patient was operated upon by the gynecologic service with a diagnosis of pelvic tumor. The infant was premature and half of its body was protruding through a defect in the fundus of the uterus. The placenta was normally attached. Both mother and baby survived.

In Case 32, the patient was a tertipara, who came to the hospital in shock. At abdominal section the placenta was found to have perforated or eroded the uterine fundus and was lying in the abdominal cavity. The 5 month fetus was dead, of course, but the mother made an uneventful recovery.

In 4 cases the ruptured uterus seems to have been associated with unusually severe uterine contractions and/or rapid labor, the so-called precipitant type of labor.

CASE 33 (1936). Colored secundipara, tertigravida, aged 24 years. The patient had a normal spontaneous delivery after a labor of 13 hours 50 minutes, during which time she had severe pains; postpartum bleeding was present. The cervical tear was repaired. Three transfusions were given; hysterectomy for tear in the lower uterine segment was performed 19 hours postpartum. She had a stormy postoperative course and returned home on the 16th day after operation. The infant lived.

CASE 34 (1937). Colored quintipara, septigravida, aged 28 years. The patient was admitted in moderate labor, the cervix dilated to 3 centimeters, station minus 1; in 1 hour the cervix was dilated to 5 centimeters in the presence of severe pains. Severe shock ensued 1 hour later, the abdomen was distended and very tender, uterus was palpated below the umbilicus. Immediate laparotomy was performed; stimulants and a transfusion of 1,000 cubic centimeters of blood were given in the operating room. The patient died 1 hour after surgery in spite of more blood and stimulants. Twin infants were stillborn.

CASE 35 (1947). Colored sextipara, septigravida, aged 32 years. The patient had been in labor for 3 hours 50 minutes during which time the fetal heart tones disappeared. Breech delivery was unattended; 2 hours 15 minutes after delivery the patient began to bleed and the uterus became doughy. For 12 hours conservative treatment was followed, then sterile vaginal examination revealed a tear in the left side of the cervix and lower uterine segment. Laparotomy was performed and 3 transfusions, sulfonamides, and penicillin were administered. The patient recovered and returned home on the 12th day after operation. The infant was stillborn.

CASE 36 (1933). White octipara, duodecigravida, aged 38 years. The patient had had severe pains for 2 hours 40 minutes before spontaneous delivery. Two hours postpartum she went into profound shock from vaginal bleeding. Vaginal examination revealed a deep tear into the lower uterine segment.

The patient died before the anticipated surgery could be performed. The infant lived.

All of the women who had a precipitant labor were multiparous (para 2, 5, 6, and 8). In 3 instances, the labor was less than 4 hours long and there are constant references in the nurses' notes to *severe* pains. Two of these mothers died (Cases 34 and 36). The cause of death apparently was shock and hemorrhage. One death occurred before transfusion could be administered or surgery could be done.

Five ruptures were due to trauma of various sorts.

CASE 37 (1932). White nullipara, primigravida, aged 21 years. The patient was admitted with a 7 months' gestation, positive Horner's sign; fetal heart tones were absent. She had a mild hypertension and pus in the urine. A 7 centimeter bag which was inserted came out spontaneously, but the patient had no pains. The bag of waters ruptured and she went into shock. Supportive treatment was given but she died of peritonitis following lower uterine segment perforation 52 hours after bag induction. Fetus and placenta were in the peritoneal cavity.

CASE 38 (1936). White nonipara, decigravida, aged 45 years. While scrubbing the floor 10½ hours before admission the patient developed severe sharp pain in the lower abdomen; vaginal bleeding and weakness followed, and she was brought to the hospital in shock. Immediate laparotomy was performed; blood was not available. She died 3 hours after admission and 1 hour after surgery. The infant was stillborn.

In Case 37 with a dead fetus of 7 months' gestation, a 7 centimeter bag was inserted. The patient died undelivered of peritonitis apparently due to a perforation of the uterine wall by the bag.

In Case 38 the patient developed symptoms while engaged in heavy household tasks. She arrived at the hospital in shock, was operated upon immediately, and expired 1 hour after surgery and 3 hours after admission. This was before the blood bank was established and the death might have been avoided had blood been readily available. Case 34 also occurred in the days preceding the blood bank.

There were 3 additional undiagnosed, traumatic ruptures not operated upon.

CASE 39 (1934). Colored tertipara, quintigravida, aged 30 years. The patient was admitted in a stupor and in labor following a severe fall downstairs while inebriated. Fetal heart tones were absent; bag of waters intact. Spontaneous delivery of the 6 month fetus occurred. Morphine sulfate and external heat (for acute alcoholic stupor with "cardiac decompensation") were administered. She died 8 hours after delivery. Postmortem examination revealed a traumatic fundal rupture. The infant was stillborn.

CASE 40 (1940). Colored tertipara, tertigravida, aged 22 years. Breech delivery occurred at home while she was unattended. Placenta was retained for 2 hours. Relatives pulled on the cord, then a physician attempted to express the placenta by Credé's method but failed, finally manual removal was carried out at home. Upon admission the patient was bleeding and had cramping pain. Three transfusions were administered for shock but the rupture was not diagnosed and no operation was attempted. She died of purulent peritonitis and endometritis 66 hours after admission with perforation of the fundus and some placental tissue in the peritoneal cavity. The infant lived.

CASE 41 (1940). White primipara, secundigravida, aged 28 years. The patient was admitted because of toxemia with a blood pressure of 170/115. Medical induction failed four times and labor was induced by stripping membranes. Bag of waters ruptured spontaneously and fetal heart tones disappeared. An attempt to convert brow failed and delivery was effected by craniotomy and cleidotomy. Total labor lasted 14 hours 50 minutes. Supportive treatment with blood transfusions, sulfanilamide intravenous fluids, and Levine suction was carried out. The rupture was not diagnosed and not operated upon. The patient died of rupture of the uterus, with infection, hemorrhage, and generalized anemia, 5 days postpartum. The infant was stillborn.

One patient, Case 39, fell down the stairs while intoxicated. Her serious condition was masked by her alcoholic stupor and she expired unoperated upon 8 hours after entrance.

In Case 40 the manual removal of the retained placenta at home resulted in death from infection.

In Case 41 comment is unnecessary regarding the trauma, shock, and bleeding associated with the failure to convert a brow except to point out that the use of blood and chemotherapy was futile because the diagnosis of rupture was not made and surgery was not done.

One of the most puzzling cases to evaluate in the entire group, as to diagnosis and treatment, is the most recent rupture of the uterus at the Cook County Hospital.

CASE 42 (1947). Colored quadripara, septigravida, aged 39 years. The patient was admitted at term with a blood pressure of 170/90. Mild Stroganoff treatment and hypertonic fluids were given.

Four days after admission medical induction with castor oil and quinine resulted in mild labor; at 10:00 p.m. on November 18, 1947, pains were at 10 minute intervals. An hour later the patient became restless; at 1:00 a.m. the blood pressure rose to 200/124 and the fetal heart tones disappeared. The cervix was effaced, dilatation 3 centimeters and station minus 2; the uterus relaxed normally between pains. At 1:50 a.m. her blood pressure was 165/105; she was co-operative at 4:30 a.m. with a pulse of 88. At 5:30 a.m. she was extremely restless and died at 5:40 a.m. undelivered. Though labor was apparently progressing normally and she was treated for hypertension, she died during labor.

This patient's clinical course suggested a nonconvulsive eclampsia and at postmortem examination it was discovered that there was also a ruptured uterus. After studying the microscopic sections, the pathology department was of the opinion that the toxemia was a large contributing factor to the cause of death.

SUMMARY

In a critical analysis of the material we find that there were 25 instances of ruptured uteri in the first 10 years (1928–1937) during which period 40,327 viable infants were delivered, an incidence of 1 in 1,613. In 11 of these patients rupture had already occurred on entrance to the hospital—2 from defective uteri, 2 from external trauma, 5 from prolonged labor (1 after receiving "black medicine"), 1 rupture after a previous classical section, and 1 after an attempted version and extraction in the home. During this decade, 11 of these patients lived and 14 died. The cause of death was: peritonitis in 7 cases; shock and hemorrhage in 5; bronchopneumonia in 1; and undiagnosed in 1 (masked by alcoholic stupor).

In the second 10 years (1938–1947), there were 17 cases of ruptured uteri, during which time 51,899 patients were delivered of viable infants, making an incidence of 1 in 3,054. In 8 of this group rupture had already occurred upon admission. Three patients had received pituitrin on the outside; 2 had had previous classical sections and 1 a previous low cervical section; 1 had had a manual removal of the placenta; 1 was a neglected transverse. In this decade, 9 women survived and 8 died. Four deaths were attributed to shock and hemorrhage and in 4 others no diagnosis was made and no operation performed.

TABLE III.—MORTALITY

	No. cases	Per cent
Mothers:		
Lived	19	45.24
Died	23	54.76
Infants:		
Lived	9	20.93
Died undelivered	5	11.63
Stillborn*	28	65.12
Neonatal death	1	2.32

*One set of twins.

The fact that the incidence was reduced by half in the latter 10 years would seem to indicate that better obstetrics is being done throughout the Chicago area. However, the infrequency of the incidence in the second period reduced the diagnostic acuity, resulting in 4 undiagnosed cases with death in each. The 7 fatalities due to peritonitis in the first decade might have been reduced in number had antibiotics and chemotherapy been available. There were 5 deaths due to shock and hemorrhage in the first decade and 4 in the second, indicating that even when adequate blood is available, ruptured uterus is an exceedingly dangerous condition and frequently fatal.

There were 13 ruptures in the upper uterine segment, 22 in the lower uterine segment, and 7 instances in which the location of the rupture was not described.

Twenty-eight women were operated upon, 18 of whom survived and 10 died. In the 6 instances in which the rupture was diagnosed, surgery was not accomplished because the course of the patients was so rapidly downhill. None of these survived. Two patients, whose condition was diagnosed, were electively not operated upon. One of these died and 1 recovered. In 6 cases in which the condition was not diagnosed and operation not performed, all patients died. The only appreciable salvage was in the group in which diagnosis was made and operation performed—64.28 per cent of these patients survived. In the group not operated upon only 1 patient (7.18 per cent) was saved. From these figures, it is evident that there is no advantage in surgical delay and very little possibility of improving the patient's condition until the cause of the hemorrhage is eliminated.

CONCLUSIONS

1. Forty-two cases of ruptured uteri (an incidence of 1:2,196) observed at the Cook County Hospital during a 20 year period, 1928–1948, inclusive, are reported and analyzed.

2. The maternal mortality was 54.76 per cent and the infant mortality was 79.07 per cent.

3. Careful observation of the intranatal patient and good obstetric practice would prevent most ruptures.

4. Diagnostic acuity, prompt surgery, plus the administration of adequate blood and judicious use of the available chemotherapeutics and antibiotics would decrease the mortality from this condition.

DEXTRAN

An Experimental Plasma Substitute

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PARTIALLY hydrolyzed dextran, administered intravenously as a 6 per cent solution in normal saline, has recently been proposed as a plasma substitute in the treatment of shock and hemorrhage. Following chemical and physiological studies at the University of Upsala under the direction of Tiselius (11), its clinical trial was first reported by Grönwall and Ingelman in 1945 (12).

Physical chemistry of dextran. Dextran is a water soluble, high molecular, natural polysaccharide first described in the nineteenth century and later found to be a common contaminant of beet sugar. It is prepared commercially by dialysis from solutions of saccharose infected by Leuconostoc mesenterioides and purified by repeated precipitation with alcohol. The dextran molecule corresponds to the formula $(C_6H_{10}O_5)_n$ with the glucose units connected by a gentiobiose (1:6 glucosidic) linkage (33). Its suggested structure consists of a long main chain of glucopyranose units with shorter side chains each composed of about 5 such units giving a postulated thickness of about 10 glucopyranose molecules (23), or about 50 angstroms. Study with the electron microscope reveals a "thread-like structure" which is usually branched and even when quite diluted seems to have no appreciable tendency to divide into smaller parts. The rough estimate of the molecular thickness of 30 to 100 angstrom units was, however, at the limit of the resolving power of the electron microscope used (17, 18). The raw material is only partially soluble in water and the resulting solutions are very viscid. It has a slow electrophoretic migration toward the positive electrode, a specific rotation of 195 degrees, and a molecular weight of many millions. By carefully controlled partial hydrolysis, a modified dextran is obtained which has a nonuniform molecular weight of between 100,000 and 200,000 and passes through capillary walls slowly. This modified dextran in a 6 per cent solution, has been proposed as a "plasma substitute" by Grönwall and coworkers. The following data describe the physical chemical properties of the dextran modified for intravenous therapy. The colloidal osmotic pressure (300 to 400 mm. H_2O) is equivalent to that of normal blood plasma; isoelectric point, *p*H 5.9 to 6.1; nitrogen content, 0.003 to 0.01 milligram per cubic centimeter; viscosity, 3 to 4 with an Oswald viscosimeter at 37 degrees Centigrade; and the buffer capacity, 6 to 7 cubic centimeters of tenth molar sodium hydroxide. The dextran sedimentation constant (determined in the ultra centrifuge) at a concentration of 0.7 per cent varies between 1.5×10^{-13} to 20×10^{-13}, and the diffusion constant varies between 0.2×10^{-7} to 8×10^{-7}.

Immunology of dextran. Grönwall and Ingelman (11) carried out a brief immunological study on dextran. Rabbits were injected with 2 cubic centimeters of 0.5 per cent dextran solution every fifth day for a period of 2 months. The sera were then tested for precipitin formation by addition of dextran solution of varying concentration. None occurred. No mention was made as to whether pure dextran or the hydrolyzed product was used in these studies. Neill and Hehre, however, had previously found that pure dextran is a serologically active polysaccharide (14, 30). They reported the formation of serologically active dextran from sucrose solution by various strains of nonhemolytic streptococci isolated from the blood of patients with subacute bacterial endocarditis, from several strains of group "H" throat streptococci, and from

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strains of Streptococcus salivarius isolated from the throat and feces. Cross-immunological relationships of the dextran so formed occurred with several strains of Leuconostoc mesenteroides and with type 2, type 12, and type 20 pneumococci. Consequently, in view of the frequent contamination of commercial sugars with leuconostoc bacteria and also with dextran itself, and because of the frequency with which pneumococcic and streptococcic infections occur, it would not be surprising if a great many people were found who would harbor antibodies capable of reacting with unhydrolyzed dextran. In support of this theory, a majority of people, skin tested by Neill and Hehre[1] with unhydrolyzed dextran, gave positive reactions in high dilution. Complete hydrolysis of the dextran molecules prevented this reaction. Whether dextran itself is capable of stimulating the formation of specific antibodies was not stated.

Physiological studies. Extensive studies on dextran and its behavior after parenteral administration have been carried out by Grönwall and Ingelman (12). Unhydrolyzed dextran in solutions of 1 to 3 per cent were injected into rabbits with resultant albuminuria. Repeated injections caused anuria and death in an unstated number of dogs. Microscopic studies revealed thrombosis of capillaries in the liver and kidneys with surrounding areas of anemic necrosis. Later, on testing solutions of partially hydrolyzed dextran, it was discovered that a dextran of medium molecular weight, in the range of 100,000 to 200,000, still retained its beneficial hemodynamic effects and could be given with safety to experimental animals. Because of the asymmetry of the dextran molecule, such molecular size was necessary in order to prevent rapid passage through the glomerular membranes.[2] This modified dextran solution was then given to rabbits and dogs as replacement therapy after experimental hemorrhage. No deleterious reactions were noticed nor was there any gross or microscopic evidence of storage in the tissues. The concentration of dextran in both the blood and urine was investigated. After administration, the plasma dextran level decreased rapidly for a few hours; then the rate of excretion became a relatively straight line curve reaching zero within 4 days. This early fall in the plasma dextran concentration is due to the rapid excretion through the glomeruli of the smaller dextran molecules, the mean molecular weight of which was calculated to be from 50,000 to 60,000.

One undesirable consequence observed following dextran was the greater tendency toward pseudoagglutination of the red blood cells and the associated increase in sedimentation velocity. Such changes bore a direct relationship to both the concentration of the dextran solution used and the size of its molecules. In vivo experiments, however, revealed that the relationships between the dextran level in the plasma and the sedimentation rate varied in different individuals. Grönwall and Ingelman concluded that although an increased sedimentation rate is, in itself, not harmful, the occurrence of injurious reactions secondary to this agglutination tendency is yet a possibility, and that a decision could "naturally not be expected before the dextran solutions have been injected in great amounts into humans, and the sedimentation reaction studied with respect to the dextran content of the blood." This same effect, of course, had been usually observed following the infusion of many other plasma substitutes such as gum arabic, pectin, and gelatin.

Clinical testing of dextran. Initial clinical testing was carried out by Grönwall and Ingelman in 1943 at the University of Upsala, and later at the Central Hospital in Örebro under the direction of G. Bohmansson and O. Wilander. Finally, large scale clinical trials were conducted at Örebro, at Stigberget's Hospital, and the Serafimer Hospital in Stockholm under Grönwall and Thorsén, and also at St. Göran's Hospital in Stockholm by Rosenkvist. Throughout this period 22 different preparations and about 1,000 bottles were used (3). Each preparation before being released had been tested on terminal cancer cases in Stigberget's Hospital.

Prior to March, 1945, many batches were discarded because of allergic and pyrogenic

[1]Personal communication.

[2]When measurement of the relative viscosity of dextran solutions was found to be a reliable test for the degree of hydrolysis, this method was employed to determine the suitability of the different preparations.

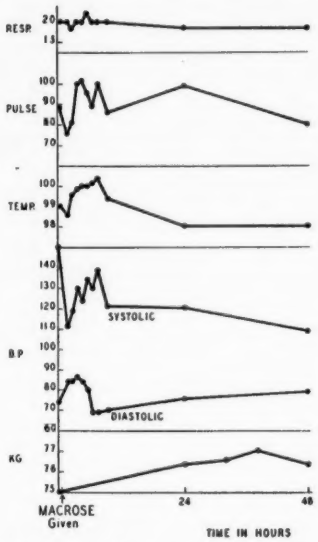


Fig. 1. Clinical response to macrose.

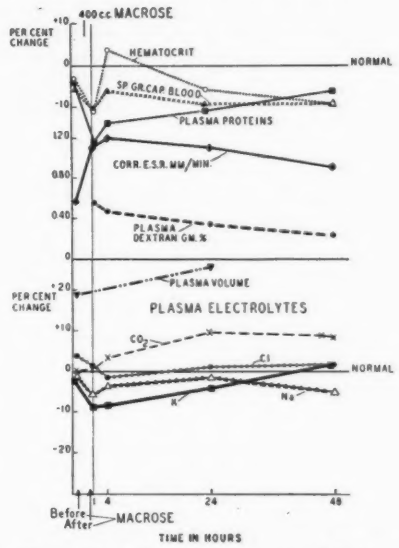


Fig. 2. Blood changes after macrose infusion.

reactions. Since then only occasional preparations have been deleted for this reason. As a result of the studies at Örebro in 1943, the composition of the dextran preparation was gradually modified: the molecular size was reduced, the viscosity was decreased from 4.5 to 5.2 to between 3 and 4, and the saline content lowered from 3 to 0.9 per cent. This new solution was introduced in 1944, and it is the preparation now employed in Sweden.

THE CLINICAL STUDIES CARRIED OUT AT THE PRESBYTERIAN HOSPITAL

Thirty chosen volunteer patients received macrose solution intravenously. One patient, a terminal cancer case, received 1000 cubic centimeters of the solution a few days prior to death. The autopsy findings in this case have not been presented in this paper because of the presence of advanced bilateral hydronephrosis consequent to the spread of the carcinomatous process from the cervix to the adnexa and the neighboring tissues and lymph nodes. This condition, unfortunately, served to obscure any renal changes which might have been found as a result of the infusion of macrose. Twenty-five patients received as much as 500 cubic centimeters each, the infusions being given by the drip method in not less than 30 minutes. No seriously ill patient was chosen. Careful clinical observations were made both during the infusion and for several days thereafter. Temperature, pulse, respiration, and blood pressure were recorded at frequent intervals. Urinalyses (routine), hematocrit, capillary specific gravity, erythrocyte sedimentation rate, blood grouping, plasma and urine dextran level, blood volume study, plasma protein level, and electrolyte studies were carried out in selected patients.

A résumé of a typical case is presented.

CASE 6. W.R., aged 16 years, male, white. Unit No. 837242. Diagnosis: ankylosis of jaw, following infection of both temporomandibular joints at the age of 3. Patient was admitted to the hospital July 16, 1946.

On July 17, a cartilage graft taken from a rib was transferred to the mandible. The postoperative course was not complicated.

On July 23, the patient was given an intravenous infusion of 500 cubic centimeters of 6.0 per cent macrose over a period of 30 minutes. There was no immediate clinical reaction to the infusion. Two hours after termination, there was a slight rise in both pulse and temperature with the latter returning to normal at the end of 24 hours (Fig. 1). The blood

TABLE I.—REACTIONS TO MACROSE SOLUTION IN CASE 6

	Premacrose	Postmacrose				
	Preliminary	1	4	24	48	72
				Hours		
Plasma dextran (grams %)		0.56	0.48	0.34	0.26	(0.2)
Corrected sedimentation rate (mm./min.)	0.57	1.10	1.20	1.10	0.90	
Hematocrit—%	45.7	41.9	48.5	44.5	42.8	43.8
Capillary blood specific gravity	1.0531	1.0508	1.0533	1.0513	1.0513	
Plasma proteins (grams %)	6.7	5.7	6.0	6.2	6.5	6.6
Plasma sodium (mEq./L)	140.0	134.0	137.0	139.5	134.8	138.1
Plasma potassium (mEq./L)	4.30	4.02	4.04	4.20	4.50	4.68
Plasma chlorides (mEq./L)	107.2	105.0	101.0	104.5	105.0	104.6
Plasma CO_2 (mEq./L)	27.0	27.2	27.8	29.5	29.2	
Plasma volume (ml.)	3773			3990		
Red cell volume (ml.)	2590			2540		
Body weight (kgm.)	75.2			76.4	76.4	76.6

pressure did not change significantly. At the end of 24 hours, a gain of 1.2 kilograms of body weight occurred whereas the plasma volume increased only 213 cubic centimeters.

There was an immediate drop and secondary rise in hematocrit, peripheral blood specific gravity, and plasma proteins (Fig. 2 and Table I) indicating a transient hemodilution. In the 24 and 48 hour specimens hemodilution was again present. The sedimentation rate doubled its initial figure and remained elevated. The dextran level fell rapidly at a uniform rate. There was no significant change in the electrolytes studied.

The patient was discharged from the hospital on July 26.

This case is presented to illustrate the various changes which took place in the blood chemistry. An interesting feature is the temporary hemoconcentration noticed at the 4 hour sample. This is what was found in those patients who reacted to the macrose solution. Those who had no reaction as a general rule showed hemodilution. Another feature is the gain in weight exhibited over a period of several days, a phenomenon noted in several patients.

SUMMARY OF CLINICAL STUDIES

1. *Clinical observations.* In several instances the blood pressure declined somewhat following the infusion; in others it was accompanied by a slight increase together with a greater rise in pulse pressure. Correspondingly small increases in temperature, pulse, and respiration were noted. In only 3 cases was the fall in blood pressure sufficiently alarming to require therapy. There were no shaking chills or spikes in fever. Flushing of the face and neck, urticaria, and a few other symptoms were experienced in several of the patients.

2. *The hematocrit.* The hematocrit determinations in 19 cases are incorporated in a scattergraph (Fig. 3). Seven of these had reactions following the administration of macrose, and separate graphs were made of these 7 patients. Hemoconcentration was observed in these reactors with definite hemodilution occurring later. Twelve nonreactors, on the other hand, showed immediate hemodilution which was maintained from 24 to 48 hours.

3. *The specific gravity of capillary blood.* Capillary blood specific gravity was measured at the bedside in 19 patients. A similar hemodilution of peripheral blood occurred in the nonreactors as was noticed in the venous blood hematocrit determinations. Similarly, those patients who had reactions showed capillary hemoconcentration which persisted for at least 4 hours. These changes are recorded by means of a scattergraph in Figure 3.

4. *Erythrocyte sedimentation rate.* The corrected erythrocyte sedimentation rate was investigated in 14 cases. These findings are de-

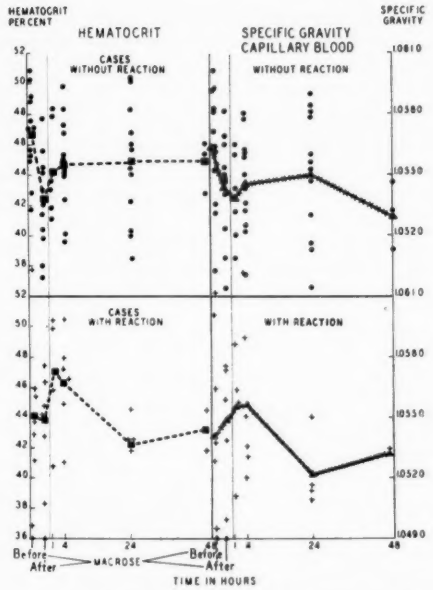


Fig. 3. Effect of macrose upon the hematocrit and specific gravity of capillary blood.

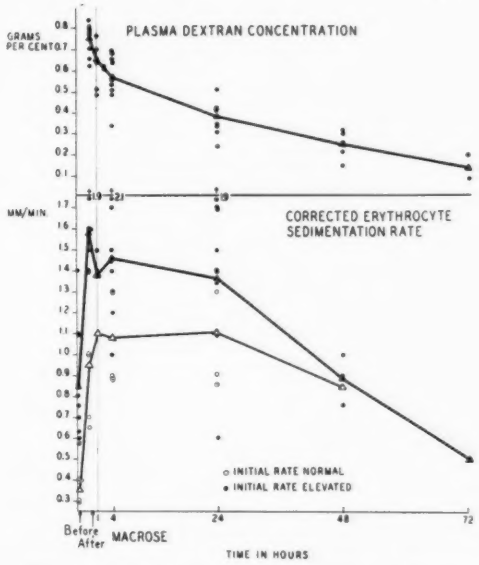


Fig. 4. Effect of macrose upon erythrocyte sedimentation rates.

picted by means of a scattergraph in Figure 4. In all there was an immediate rise in the sedimentation velocity. The subsequent decline in the erythrocyte sedimentation rate to the original values could not be definitely correlated with the decrease in the plasma dextran level. An in vitro experiment demonstrated, however, that with any given blood sample a rough correlation between the erythrocyte sedimentation velocity and the concentration of dextran did exist (Fig. 5).

5. Plasma dextran levels. Plasma dextran determinations were carried out in 12 cases. The highest levels were found in the 15 minute specimens and in 1 case it was 0.84 gram per cent. The concentrations fell rapidly for the first few hours and then assumed a more gradual but uniform rate of fall. At 24 hours, the plasma dextran was approximately one-half of the postinfusion level (Fig. 4). By the third day, a very low concentration of dextran remained in the plasma.

6. Blood grouping. The blood groups were typed before and immediately after the macrose infusion in 3 cases, whole blood on slides and cell suspensions in saline being used. No difficulty was encountered in obtaining the correct blood group.

7. Urinalyses. Urinary dextran determinations, although not reliable quantitatively, indicated qualitatively that dextran passed into the urine during the first 24 hours but very little was excreted during the second 24 hour period in 3 of the intensively studied cases. Routine urinalyses before and for sev-

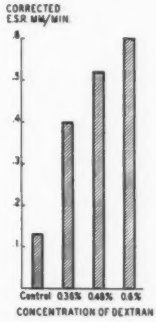


Fig. 5. Effect of varying concentration of dextran on sedimentation rate in vitro.

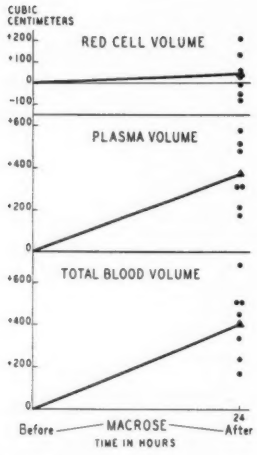


Fig. 6. Blood volume after macrose.

eral days after the infusion revealed no important changes.

8. Plasma volume. Plasma volume was measured in 7 cases. These values are recorded in graph form in Figure 6. At the end of 24 hours the plasma volume of these 7 cases was an average of 370 cubic centimeters greater than it had been prior to the infusions. None of these patients had had reactions.

9. Plasma proteins. Plasma proteins were analyzed in 6 patients. These are shown in Figure 7. The drop in plasma proteins roughly parallels the hemodilution as observed by means of the hematocrit and capillary blood specific gravity.

10. Electrolytes. In 7 cases the concentration of sodium, potassium, chlorides, and bicarbonate ions were determined before and at intervals after the infusion of macrose. Both the sodium and potassium ions decreased, the latter more than the former due to the sodium contained in the infusion. After the first hour the sodium concentration continued to fall and that of the potassium ion to rise. At the 48 hour postinfusion sample, the values had returned to normal.

The chlorides were little affected. Immediately after the macrose there was a small rise due, no doubt, to the chloride ion in the infusion. A slight acidosis existed in the cases studied. Following macrose, this condition was improved by the end of the 4 hour period. On the whole the variations in the electrolytes in these 7 patients was not significant (Fig. 8). More such studies are needed.

11. Reactions to macrose. Of the 30 patients given infusions of macrose, there were 10 who had definite reactions. Four were females and 6 males. Six received the full 500 cubic centimeters of the 6 per cent macrose solution. In 4 cases, the infusion was stopped after 400 cubic centimeters, 20 cubic centimeters, 400 cubic centimeters, and 75 cubic centimeters, respectively, because of the advent of reactions. These reactions have been divided roughly into 3 different groups: (1) skin reaction (flushing, pruritus, and urticaria), 6 cases; (2) circulatory reaction (cyanosis and fall in blood pressure), 3 cases; (3) asthmatic reactions, 1 case. Nineteen of the 20 remaining cases received the full 500 cubic centimeters of macrose solution intravenously without symptoms. The twentieth case received 1000 cubic centimeters, (500 c.c. on 2 successive days). Those patients who had reactions of one kind or another also had significant changes in the temperature, pulse, respiration, or blood pressure. Other symptoms which may or may not be significant are headache, 3 cases; drowsiness, 3 cases; and restlessness, 2 cases. In 7 of the 10 who had reactions, and in whom hematocrits and peripheral whole blood specific gravity tests were carried out, hemoconcentration was observed. Hemodilution, on the other hand, was the rule for all those who did not have reactions. In the nonreactors, blood studies were carried out in 12 of the 20 and hemodilution in some degree was noted in each. The changes in the hematocrit and in the specific gravity of capillary blood of both those with reactions and those without reactions are presented in Figure 3. Average values are recorded for both those patients who had reactions and those who did not. As the groups presented are statistically small, and as the preinfusion values of the hematocrits and specific gravity determinations varied widely from patient to patient, this graph is open to criticism. On the other hand, the general trend observed is what is found on individual analysis of any of the different cases. As allergic manifestations character-

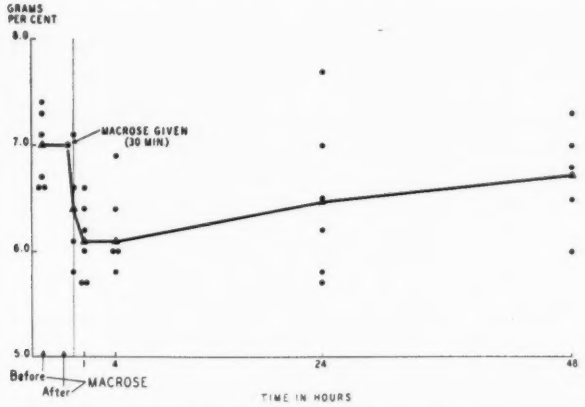


Fig. 7. Changes in plasma proteins (biuret) after macrose.

istically show hemoconcentration, it is likely that the reactions observed are related and are either of anaphylactic or anaphylactoid nature.[1]

A résumé of Case 30 is presented. This patient experienced a severe asthmatic reaction.

J.M., aged 21 years, male, white, Unit No. 669788. Diagnosis: recurrent osteomyelitis. Patient was admitted to the hospital July 22, 1946. Discharged August 18, 1946.

This patient had developed an osteomyelitis of the right femur and left radius in March, 1942 and had subsequently been admitted to the hospital on four separate occasions for treatment. A fifth admission in January, 1944 was because of bronchial asthma, influenza, and bronchopneumonia. Sputum cultures at this time were positive for Pneumococcus type 20. His past history was irrelevant except for "hives" as an infant and an unsubstantiated history of hay fever for two summers. Physical examination showed a healthy young male with positive findings limited to the right hip and the left wrist. Culture of pus from sinus of right hip was positive for Staphylococcus aureus.

On July 22, incision and drainage of an abscess of the right hip was carried out. His postoperative condition was good. On July 24, upon completion of the routine preliminary control tests, an infusion of 6 per cent macrose was started at 9:05 a.m.[2] The rate of the infusion was approximately 60 drops per minute. Within a very few minutes, the patient's face and neck became flushed and he began to cough repeatedly. Auscultation of the chest revealed a few expiratory wheezes. The macrose infusion was stopped immediately (not more than 20 c.c. had been given), and the patient was given 3 minims of 1:1000 adrenalin by hypodermic. When it was seen that the patient was becoming increasingly dyspneic and cyanotic, oxygen by mask was instituted and a second 3 minims of adrenalin was given. Following this therapy there was a gradual lessening of the dyspnea so that one hour later all symptoms had disappeared.

[1]Sterile infusion sets were used throughout. Baxter Number 18-R, Number 11-R, and Number 8 sterile tube and needle sets were used. Reactions were obtained with each of the three different recipient sets.

[2]Bottle No. 33, lot No. 15521, control no. B2-2, infusion set Baxter No. 18-R.

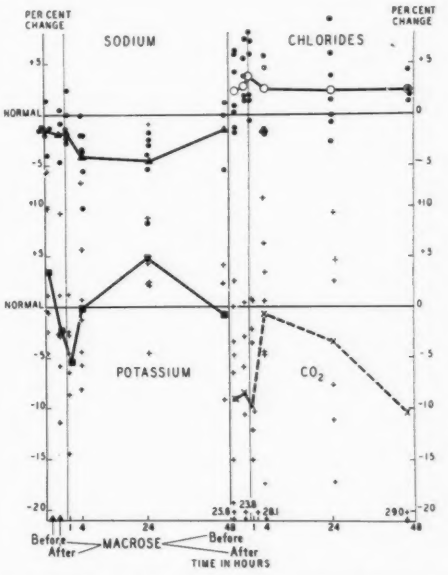


Fig. 8. Effect of macrose upon the plasma electrolytes.

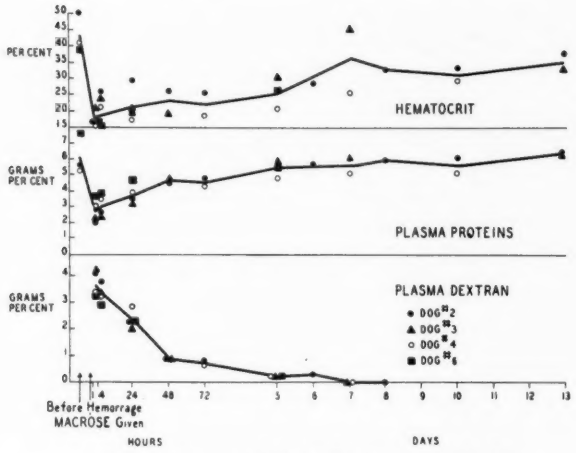


Fig. 9. Macrose infusion following exsanguination.

Since he received only 20 cubic centimeters of macrose, no significant concentration of dextran in the plasma was detected. The sedimentation rate did not change until 24 hours later when a significant rise occurred. There was an immediate rise in the hematocrit followed by a fall back to normal in the 4 hour specimen. There occurred no appreciable change in the specific gravity of peripheral blood or the plasma protein concentration. Blood volume before and 24 hours after macrose was little altered. The variations in electrolytes were only minor. Plasma sodium fell during the first 4 hours and returned to normal at the end of 24 hours. There was a slight increase in the plasma potassium between the sample taken at the 4 and 24 hour periods. There was no alteration of body weight during the first day. The patient was discharged from the hospital on the 15th day after operation. No late sequelae were noted either in the hospital or in the out-patient department.

PATHOLOGICAL STUDIES AT THE PRESBYTERIAN HOSPITAL

Resuscitation of dogs with macrose, and subsequent autopsy findings.

Six dogs were bled an estimated 50 per cent of their blood volume and were immediately given an equal volume of 6 per cent macrose. The experimental procedure was similar both to that used by Ivy and his coworkers in 1943, and to the method adopted by Buttle, Kekwick, and Schweitzer in cats, 1940.

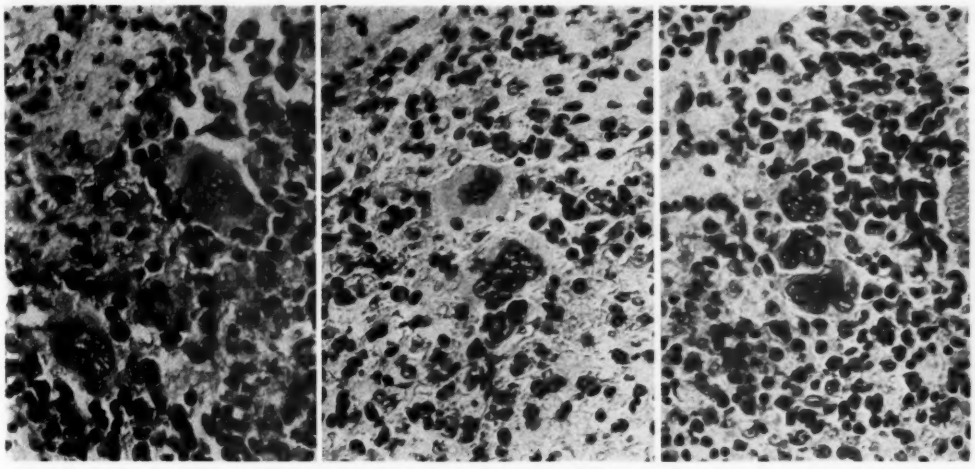
Under procaine local anesthesia, the jugular (internal) vein and common carotid artery on one side were exposed. The carotid artery was then cannulated and the blood allowed to flow into a graduated cylinder until the animal stopped breathing, or until the arterial bleeding was reduced to a slow drip. When this state was reached, the animals were given a rapid infusion into the internal jugular vein of a volume of macrose equal to that of the blood lost.

At 1, 4, and 24 hours following the infusion and then every other day up to the 13th day, jugular vein blood was secured for hematocrit, plasma dextran, protein, and specific gravity.

Results: These 6 dogs lost on the average, as a result of their bleeding, 42 per cent of their total blood volume.[1] After receiving 400 to 500 cubic centimeters of macrose, they were all able to walk unaided. In fact the clinical response of the dogs to the infusion was quite dramatic. Of the 6 dogs, only 1 died. This animal had pneumonia, responded well to the infusion but then gradually weakened, and after exhibiting increasing drowsiness, was found dead in his cage 3 hours after the infusion.

The blood studies on 4 of these dogs are present in Figure 9. The hematocrit dropped to a low of 15.8 per cent and rose very slowly to near the initial level at the end of 2 weeks. The plasma proteins had an equally sharp fall, but recovered more quickly with some

[1]The calculated blood volumes were determined by the identical method used by Ivy et al. (19), i.e., 9.5 per cent of body weight.



a b c

Fig. 10. a, b, c (dogs 2, 4, 6), 14 days, 11 days, and 5 days after infusions. Spleens. There is moderate reticuloendothelial hyperplasia with the formation of multinucleated giant cells. ×325.

figures near the preliminary level at the end of 1 week. The plasma dextran levels decreased from levels as high as 4.1 grams per cent immediately after the infusion to a very low level by the fifth and sixth days. No marked change was found in plasma specific gravity determinations carried out before and for several days after the infusion.

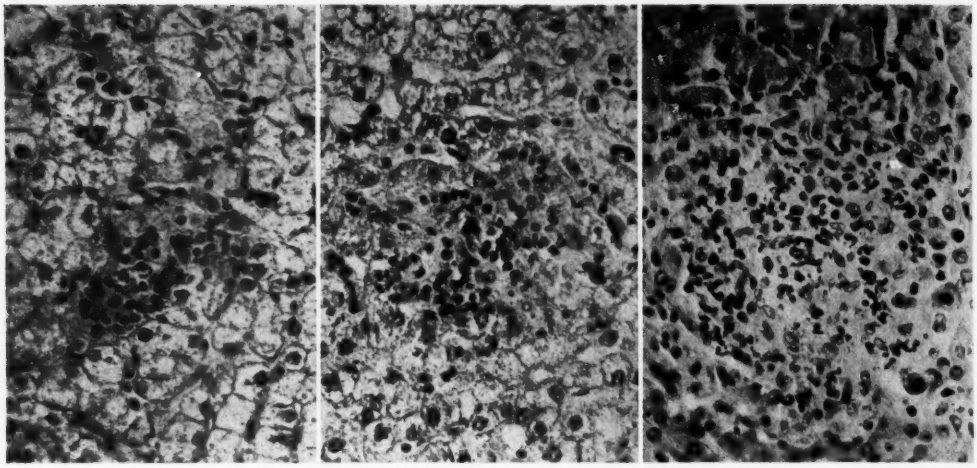
Autopsy studies. Each of the 6 dogs was subjected to autopsy and microscopic sections were prepared. Dog 5 with pneumonia died 3 hours after the infusion. Dogs 1, 2, 3, 4, and 6 appeared strong and in general good health and were sacrificed on the 19th, 14th, 13th, 11th, and 5th days, respectively, after the infusions.

Autopsy studies on dog 5 revealed no gross changes of interest other than a general anemia and pallor of all the tissues, and a nodular consolidation of the right middle and right lower lobes and of the left lower lobe. Histological studies confirmed the diagnosis of a diffuse lobular pneumonia of severe degree.

Autopsies on the other 5 dogs were carried out at the same time. The tissues were grossly normal in appearance and the organs were of normal size and weight. There was no evidence of edema, and no increases in peritoneal, pericardial, or pleural fluids. General pallor of the tissues was evident. Specimens for histological study were taken from the livers, spleens, kidneys, and lungs.

Microscopic examination.[1] Sections of lungs revealed no change. The spleens of all 5 dogs (Fig. 10) showed evidence of moderate reticuloendothelial hyperplasia together with the formation of multinucleated giant cells of epithelioid type. These cells resembled megakaryocytes. The livers of dogs 3 and 4 presented multiple small areas of midzonal focal necrosis (Fig. 11). The liver of dog 1 exhibited several small focal collections of mononuclear cells without evidence of necrosis. The kidneys of dog 3 appeared normal with exception of slight dilatation of some of the tubules and some of Bowman's capsules. The kidneys of the other 4 dogs (Figs. 12, 13) exhibited varying degrees of focal glomerular and tubular damage with atrophy, necrosis, and some epithelial proliferation of the glomerular tufts, minimal hyalinization of the loops, edema, vacuolization, fatty and colloidal degeneration of tubular cells, and dilatation of the tubules and of the Bowman's capsules. Less than 10 per cent of the total glomeruli and tubules were involved by this process. Eosinophilic hyaline débris and clumps of pinkish-purple amorphous material arranged sometimes in concentric rings were found within a number of glomeruli. Similar débris and clumps of epithelial cells in various stages of necrosis and calcification were present in some of the collecting tubules. Special stains for iron and calcium were employed, and they

[1]The microscopic sections are from the Department of Surgical Pathology under the direction of Dr. Arthur P. Stout, Professor of Surgery.



a b c

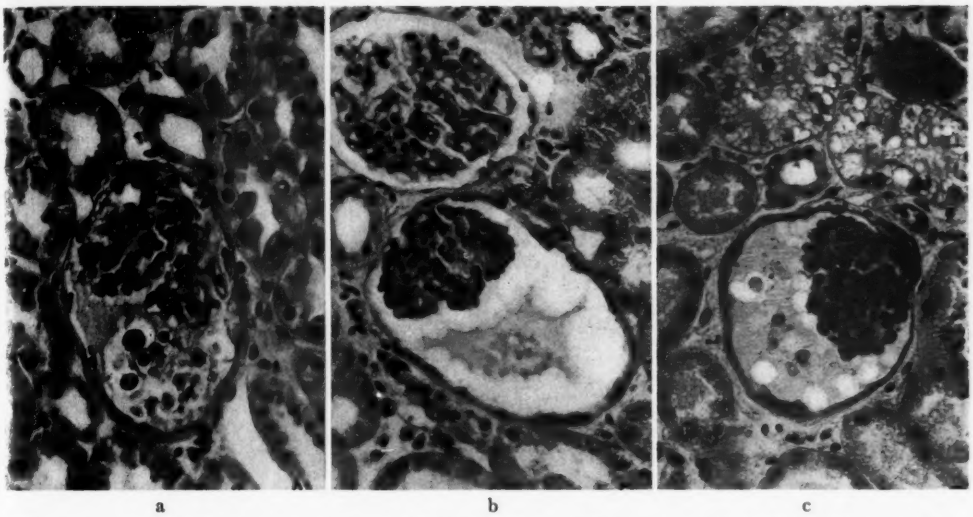
Fig. 11. Dog 1. a, Nineteen days after infusion. Liver. In the center of the field there is a collection of mononuclear cells together with a few polymorphonuclear leucocytes. The hepatic cells show extensive glycogen storage. ×325. b, Dog 3. Thirteen days after infusion. Liver. Focal midzonal necrosis. The hepatic cells show extensive glycogen storage. ×325. c, Dog 4. Eleven days after infusion. Liver. Focal midzonal necrosis. ×325.

confirmed the presence of these substances in this amorphous débris. Both the lower and upper portions of the nephron were involved in this degenerative process. The fatty degeneration, however, involved only the proximal convoluted tubule. That the glomerular damage was of recent date was evidenced by the lack of organization or scar formation.

DISCUSSION

The attributes of an ideal plasma substitute have been summarized by Amberson, Moon, Cohn, Scudder (38), and others. Briefly such an agent should have: A colloidal osmotic pressure of plasma proteins (300 to 400 mm. of water); large molecular size with molecules of symmetrical shape; low viscosity; chemical stability in the wet and dried states; an ionic strength of physiological proportions; a high buffer capacity; and a *p*H near that of blood. It should be nontoxic, nonantigenic, easily metabolized, not retained as a foreign body, and possess the power of restoring protoplasmic irritability. Finally it should withstand sterilization, transportation, and storage without deteriorization. A 6 per cent solution of partially hydrolyzed dextran fulfills many of these strict prerequisites but not all. One striking deficiency is the long and highly asymmetrical molecular structure.

One harmful effect of this asymmetry is the greatly increased erythrocyte sedimentation velocity observed in all the patients given macrose. This effect is a characteristic of many macromolecular substances. In 1918, Fåhraeus demonstrated that the sedimentation rate was correlated with changes in certain plasma constituents especially fibrinogen and globulin, and in 1921 Fåhraeus published an exhaustive historical review and experimental study on the suspension stability of the blood, and reported that gum arabic and other macromolecular substances produced pseudoagglutination and consequently increased the sedimentation velocity. This work, together with that of Karsner and Hanzlik and of others, has been augmented in recent years so that now Meyer and associates in 1945 believe that probably all highly asymmetrical molecules of large size will cause an increase in the sedimentation rate when added to normal blood. The reasons for this phenomenon are not definitely known. However, it has been observed in many instances to be reversible and it also has been observed that chemical and enzymatic breakdown and depolymerization of these high molecular weight substances will prevent any such increase in the agglutinabil-



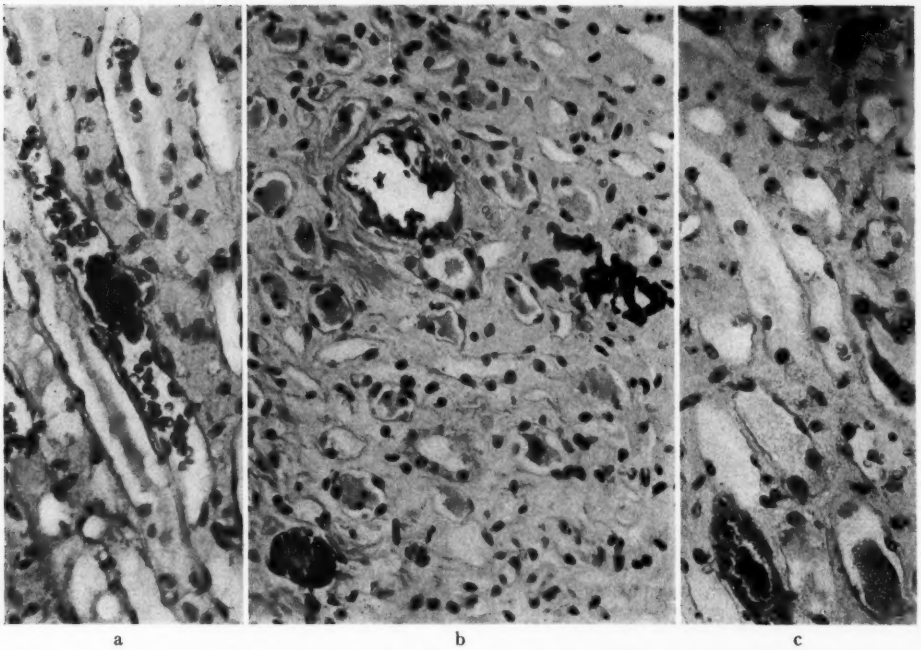
a b c

Fig. 12. a, Dog 1. Nineteen days after the infusion. Cortex of kidney showing shrunken glomerular tuft and dilated Bowman's capsule. The lumen of the glomerulus contains calcified masses of foreign material and eosinophilic débris. ×325. b, Dog 2. Fourteen days after infusion. Cortex of kidney showing shrunken glomerular tuft with pyknotic nuclei. The glomerular space contains eosinophilic débris and Bowman's capsule is dilated. ×325. c, Dog 4. Eleven days after infusion. Cortex of kidney showing shrunken necrotic glomerular tuft, dilatation of the capsule, and calcium containing débris within the lumen. Note the ringed appearance of the foreign bodies in the glomerular space. The tubules show fatty degeneration. ×325.

ity of red blood cells (28). Nungester and Klein reported that the addition of type 3 Pneumococcus polysaccharide to normal blood would also increase the sedimentation rate and that type specific antiserum would abolish this effect. As dextrans are serologically active bacterial products (14), we now have a second known bacterial product which will increase the erythrocyte sedimentation rate. It is likely that many such substances will be found in the future.

The extent to which this macromolecular substance of asymmetrical shape is stored in the body and its catabolism are not yet known. In vitro experiments have thus far revealed only a slow reduction with starch splitting enzymes (12), and as no evidences of accumulation have heretofore been detected in experimental animals, it is believed that a gradual enzymatic destruction of the molecule does take place. Slow excretion or breakdown of the dextran molecules is, of course, desirable. In the treatment of the shock state one is primarily interested in restoring the circulating blood volume to normal, and in maintaining it at that level until the body is able to compensate for the loss of fluid volume through its own compensatory mechanisms. It is only when there is severe reduction in blood volume, tissue anoxia, and capillary damage that prompt therapy is mandatory employing plasma, or a plasma substitute until blood is available. Such aid is needed for only a short time and as a rule for not more than 12 hours. It is known that regeneration or restoration of plasma proteins after blood loss takes place rapidly if shock is not present (25). As much as 10 to 29 per cent of the normal plasma proteins enters the circulation in the first 12 hours and a similar amount during the second 12 hours (8). Our own experiments with dogs discussed in this paper tend to confirm these observations.

Is partially hydrolyzed dextran toxic? The histological findings observed in a series of 6 dogs who received macrose solution intravenously have been described. Some of these changes are not reversible but were minimal,



a b c

Fig. 13. a, Dog 1. Nineteen days after the infusion. Medulla of kidney showing cellular cast within the lumen of a tubule. Calcified débris is present within the tubule in the center of the photograph. ×325. b, Dog 4. Eleven days after infusion. Medulla of kidney. There is calcified débris within the lumens of three collecting tubules. The tubular epithelium is necrotic. On the right, cellular outlines can be distinguished within the partially calcified mass. ×325. c, Dog 6. Five days after infusion. This photomicrograph of the medulla of the kidney shows tubular degeneration with calcified débris in the lumen. At the top of the photomicrograph, a tubule is seen which contains numerous erythrocytes as well as calcified débris. ×325.

and only focal in nature. In comparison with the Swedish group who observed no pathologic alterations in the tissues, the amount of macrose solution used in our animals was relatively high. These changes are somewhat similar to those found by others investigating several colloidal plasma substitutes.

Hueper contends that certain macromolecular substances employed as plasma substitutes (acacia, polyvinyl alcohol, pectin) are pathogenic when administered intravenously. He has presented evidence revealing organic changes to be frequently found, and characterized by: storage in the reticuloendothelial system, degenerative vascular changes often identical with atheromatous or arteriosclerotic lesions, and degenerative and proliferative changes in the glomeruli. Popper and associates have also observed pathological changes following the administration of macromolecular substances.

Is partially hydrolyzed dextran antigenic? This question cannot be answered at this time; the reader, however, is referred to the experimental work of Neill and Hehre. Our own experience, which includes 10 reactions in a series of 30 cases, leads us to believe that these reactions were of allergic nature. Whether chemical modification of the dextran solution will prevent any further reactions of this type we do not know, but in view of the success had with this plasma substitute abroad, we feel that this is likely.

As to the cause of the reactions observed, three possibilities are offered: (1) They were the result of protein impurities present in the solution. (2) The partially hydrolyzed dextran molecules themselves possessed definite

serological activity. (3) The reactions were not anaphylactic but anaphylactoid in nature, and consequent to the cellular damage produced by the action of this macromolecular substance. It is not believed that the presence of protein impurities was likely because the nitrogen content of the bottle of macrose used in Case 30 was determined in duplicate and assayed to be only 0.0051 and 0.0050 milligram per cubic centimeter. Aerobic and anaerobic cultures were also carried out on 6 bottles of macrose, 2 of which caused allergic manifestations. These were negative for leuconostoc and other bacteria. By way of further analysis, blood sera from 4 patients who had received dextran were then sent to Neill and Hehre for study. These sera were taken 7, 11, 24, and 27 days after the macrose infusions. Precipitin reactions were carried out with negative results.[1] A culture of Staphylococcus aureus from patient 30 with osteomyelitis was also submitted to Neill and Hehre; it was found not to produce dextran. Patient 30, who suffered a severe and immediate asthmatic reaction to the dextran infusion, had been admitted to the hospital in January, 1944, with a diagnosis of bronchial asthma, and bronchopneumonia. His sputum cultures at that time revealed large numbers of Pneumococcus type 20. This is of interest as Neill and Hehre had shown immunological relationships between Pneumococcus type 20 and dextran. No similar relationship was traced in any of the other reactors.

In 3 of these cases the more common symptoms and manifestations of allergy (such as asthma, urticaria) were absent. Instead, they presented signs of cardiovascular collapse with a drop in blood pressure, cyanosis, and in 1 patient definite lowering of temperature of the extremities. All required supportive therapy: oxygen by mask and 5 per cent glucose and saline. Hemoconcentration was present in each. Plasma sodium, potassium, chlorides, and carbon dioxide determinations were carried out in Case 19 but no significant changes were found.

It is likely that here again we have an anaphylactic or anaphlyactoid reaction but with clinical involvement of a different tissue. It is of interest to recall, at this point, the work of Hanzlík and Karsner who in 1920 had shown that many colloidal substances on intravenous administration will give rise to anaphylactoid phenomena of circulatory and respiratory nature.

Cyanosis was an interesting phenomenon noticed in several of these cases. This discoloration was at first discounted because blood volume studies by means of the Evans' blue dye technique were being carried out. However, in other patients who did not have these blood volume measurements, cyanosis of mild degree was also observed. In all cases blood samples drawn after the macrose infusion appeared darker than did the preliminary blood specimen. Shaking this blood would lighten the color, but later, after settling had taken place, the blood would again become darker than its control. In patient 19 where cyanosis was an important feature together with vascular collapse, an analysis of the oxygen carrying capacity revealed a normal value of 18.9 volumes per cent. In carrying out this analysis, however, the chemist remarked that it took abnormally long for the blood to become completely saturated. Such reduction in the speed of oxygenation of the erythrocytes has been observed both in the human and in the experimental animal after polyvinyl alcohol, methyl cellulose, acacia, and gelatin (6, 19, 26). A similar discoloration of blood was noticed when Nungester added Pneumococcus polysaccharide to blood. Christie, Phatak, and Olney reported in 1935 that although the carbon dioxide content of the blood after administration of acacia decreased proportionately with the hemodilution as observed by Hanzlik, the oxygen content fell at the end of 1 hour to a more marked degree than would be expected, and that this effect persisted for more than 24 hours. This effect has been attributed by Hueper to the formation of surface films of the macromolecular matter on the erythrocytes with resulting impairment of the gaseous interchange across the interface.

Two other possible reasons for the cyanosis in these cases are related to the well known effect of macromolecular substances in increasing the conglutination of erythrocytes as first noted by Hanzlik and Karsner in 1920 and

[1]Personal communication.

Fåhraeus in 1921. (1) This conglutination could result in a decreased functioning surface area for use in the interchange of gases. (2) It also might result in slowing and obstructing the flow of blood in the smaller capillaries. Fåhraeus, Parkins, Hopps, and Youngner and Nungester, all have reported the microscopic visualization of capillary blood flow following the administration of various colloidal solutions and plasma substitutes. Each of these authors noted that pseudoagglutination of red blood cells took place. Their observations, however, show no unanimity of opinion as to whether or not slowing of the blood flow actually did take place as a result of this agglutination.

CONCLUSIONS

1. Thirty volunteer patients at the Presbyterian Hospital were given infusions of macrose, a 6 per cent solution of partially hydrolyzed dextran. The lot number of the solution used in each case was B2-2. Ten of these patients had reactions of either anaphylactic or anaphylactoid nature. A striking increase in sedimentation rate was noted in all. These reactions are believed to be specifically related to the molecular structure of the dextran molecule and its concentration in the plasma. A possibility exists that some undiscovered impurity was present in this solution, but in view of the work of Neill and Hehre we feel that the weight of evidence incriminates the dextran molecule itself.

2. No great changes in the levels of the various blood electrolytes were found following the administration of dextran solution. Studies were carried out on the plasma sodium, potassium, chlorides, and carbon dioxide.

3. Satisfactory hemodilution and increase in plasma volume was noted after macrose. This effect persisted for a period of at least 24 hours.

4. Plasma proteins regenerated rapidly following exsanguination and replacement of the blood loss with macrose solution.

5. Autopsy studies on 6 dogs who received macrose, Lot B2-2, in large quantities revealed focal degenerative lesions in the livers and kidneys, and minimal reticuloendothelial hyperplasia in the spleens.

NOTE.—The clinical testing of macrose solution was carried out at The Presbyterian Hospital in the City of New York on the Second Surgical Division of the late Dr. Hugh Auchincloss, between June 25, 1946, and July 25, 1946. In all, 30 patients received macrose solution intravenously. Because of the severity of the reaction encountered, in Case 30 with recurrent osteomyelitis, it was considered unwise to continue the clinical testing of this batch of solution beyond this date.

Methods for determinations

The *hematocrits* were centrifuged in Sanford-Magath tubes for one hour at 3,000 r.p.m. using heparin as an anticoagulant (36).

The *sedimentation rates* were determined by the Rourke-Ernstene method (35).

The *concentration of macrose in plasma* was determined by the method of Harry Hint, Pharmacia, Ltd. (unpublished) as adapted by Margaret E. Smith for the Evelyn photoelectric colorimeter.

The *whole blood specific gravity determinations* were obtained by the method described by Barbour and Hamilton (2).

The *plasma volume determinations* were done by the dye (T-1824 or Evans blue) dilution method of Gregersen (10).

The *plasma proteins* were determined by the biuret method of Kingsley (21) as modified by Mehl (27) and adapted for use with the Evelyn photoelectric colorimeter.

The *carbon dioxide combining power* was determined by the method of Van Slyke and Neill (39).

The *plasma chlorides* were determined by the potentiometric titration method (22, 24).

The *plasma sodium levels* were obtained by the gravimetric method of Butler and Tuthill (4).

The *potassium analyses* were done by a modification of the argenticobaltinitrite method (37).

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CYTOLOGIC EXAMINATION OF SPUTUM AND BRONCHIAL SECRETIONS IN DIAGNOSIS OF BRONCHOGENIC CARCINOMA

IT is becoming evident that primary carcinoma of the lung is a much commoner lesion than has been recognized in the past. In part, this results from the inaccessibility of a good portion of the bronchial tree to ordinary bronchoscopic methods. It has been claimed that the cytologic examination of sputum and bronchial secretions results in many more definitive diagnoses of cancer of the lung.

The rationale for the examination of secretions throughout the body for carcinoma depends on the exfoliation of carcinoma cells from malignant tumors having a free surface. Cancer cells differ from normal cells by having large nuclei, in irregularity in size of cell and nucleus, altered nuclear-cytoplasmic ratio, hyperchromatism and large nucleoli. This principle was noted by Papanicolaou[1] in 1928 while working with vaginal secretions.

Up to 1935 only an occasional diagnosis of carcinoma of the lung had been made by the examination of the sputum and usually then, when a fragment of tumor was identified in the sputum. In 1935, Dudgeon and Wrigley[2] devised a special technique for the cytologic examination of sputum. This method involved the fixation of wet films in Schaudinn's solution and staining with hematoxylin and eosin. By this means they were able to demonstrate malignant cells in 68 per cent of 58 cases of proved bronchogenic carcinoma. Barrett[3] in 1938 and Gowar[4] in 1943 reported positive results of examination of sputum in 68 and 64 per cent of cases, respectively. A monograph was written on the subject by Wandall[5] of Copenhagen in 1944. He found that the sputum contained cancer cells in 84 per cent of 100 cases of proved bronchogenic carcinoma. Results of bronchoscopic biopsy were positive in 55 per cent of patients who were studied in this manner. A study of secretions removed bronchoscopically from patients was carried out by Herbut and Clerf[6] in 1946. They reported positive results in 82.4 per cent of 57 cases of proved bronchogenic carcinoma. It was felt by Herbut and Clerf that sputum was an inaccurate and too time-consuming material for study. Papanicolaou[7] in 1946 reported positive results from the examination

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of sputum in 88 per cent of 25 cases of proved bronchogenic carcinoma. Woolner and McDonald examined sputum, bronchial secretions and bronchial washings and have felt that all materials should be utilized. During this time they have made a positive diagnosis of cancer cells in approximately 325 cases[1–6]. The stain which they have employed has been hematoxylin and eosin.

Bronchial washings are obtained by instilling 5 cubic centimeters of saline solution into the affected bronchus and recovering the material. This can be centrifuged and the sediment stained after smearing. Bronchial secretions as obtained through the bronchoscope can be handled in comparable fashion to sputum. The advantages of bronchial secretions and bronchial washings are that material can be more or less selectively obtained from the affected bronchus. The specimen is undiluted with extraneous material such as one obtains from the mouth and other parts of the lung. It has the further advantage that it makes possible the examination of patients who cannot produce sputum, and it takes less time to examine than does sputum. The most obvious advantage of sputum is its ease of collection and the facility with which one can obtain multiple specimens for examination. In cytologic work, examination of multiple specimens is very important.

The authors have employed the smear technique rather than placing the material in a paraffin block and sectioning. Probably the most important part of the technique is the inclusion of Papanicolaou's suggestion; namely, wet fixation of the smears in a fixative (50 per cent ether and 50 per cent of 95 per cent ethyl alcohol). It becomes very obvious that considerable experience is necessary to run a cytologic laboratory. The average time required for the examination of a single smear of sputum is ten minutes; five smears are examined from each sputum and in cases in which cancer cells cannot be found, three specimens of sputum have been examined, with a total average examination time of two and one-half hours. If the patient is ambulatory, he is given 30 cubic centimeters of 95 per cent ethyl alcohol in a bottle and the sputum is collected in this fixative. This obviates the necessity of obtaining fresh material, which is very difficult, particularly on an out-patient service.

In Woolner and McDonald's first 300 positive specimens of sputum and bronchial secretions, they had 4 proved false positives, 3 of which occurred in the first 150 cases. The vast majority of positive cases proved to be primary bronchogenic carcinoma. They demonstrated that cells were exfoliated in many cases of alveolar carcinoma, but only occasionally in metastatic carcinoma and never in adenoma of the bronchus. Occasionally, positive cells could be recovered in cases of cancer of the larynx and of the esophagus.

Of those patients in this group who underwent bronchoscopy, positive tissue was obtained in 55 per cent of incidences. It would appear, therefore, that the cytologic examination of sputum and bronchial secretions is of definite aid in the diagnosis of primary carcinoma of the lung.

In an endeavor to determine the false negative error, they investigated a group of 588 cases in which cytologic examination was carried out during the time 100 positive re-

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sults were obtained. The final clinical diagnosis in this group of cases proved to be bronchogenic carcinoma, 147 cases; indeterminate, probably nonneoplastic, 39 cases; probably metastatic carcinoma, 30 cases; adenoma of the bronchus, 5 cases; nonneoplastic pulmonary disease, 367 cases. The smear technique thus resulted in 68 per cent positive results, a figure which would closely approximate that of the English workers. It would appear that the false positive error should be less than 2 per cent while the false negative error is about 30 per cent. Under such circumstances, the method will prove to be a valuable diagnostic aid in cases of cancer of the lung.

JOHN R. MCDONALD.
LEWIS B. WOOLNER.

THE RISK OF SURGICAL OPERATION

An inevitable question to be dealt with whenever surgical operation is being considered is its risk. The patient may ask directly how dangerous the operation is, the point may be covered implicitly in the discussion, or the patient may leave the question entirely to the surgeon's best judgment. The risk of operation is frequently difficult to weigh; the danger must be balanced against the probable benefit; an intensely personal relationship between surgeon and patient is involved, yet other members of the patient's family, other possible patients and other physicians may be concerned in the decision. All these points create an intellectual challenge of the first order for the surgeon, and the challenge is just as real in relation to the surgical treatment of advancing and otherwise incurable disease as in "elective" operations.

In the first place it is obvious that the patient consulting a particular surgeon is concerned with the risk of operation as performed by that particular surgeon, with his particular skill and facilities. Morbidity or mortality figures from other clinics may have little bearing on the case at hand. In this connection we should note a source of error prevalent in medical graduate teaching. The error might be called a confusion of identity. For example, an experienced gynecological surgeon may report from a large clinic that the mortality rate of total hysterectomy is only a fraction of a per cent higher than that of supravaginal hysterectomy, and further may show that the incidence of carcinoma in the remaining vaginal stump more than nullifies the slight additional risk of total hysterectomy. Or, a surgeon with a mortality rate of one per cent for subtotal gastric resection for supposedly benign gastric ulcer may report an incidence of 10 per cent for carcinoma in the removed specimens, and conclude that all chronic gastric ulcers should be treated by subtotal gastric resection. For the two surgeons the facts are present and the conclusions valid. However, when the conclusions are passed with the weight of authority to surgeons throughout the country serious harm may be done. The point may be missed that sound practice for a specialist in a large teaching center may not be safe practice for the average surgeon.

In the next place, the patient is concerned with the operative risk for him as an individual, and although he may be interested in the results his surgeon achieved in the last one hundred cases the significant point is the personal factor. What is his physiological reserve and how do his age and associated diseases affect his chances? How does his obesity, hypertension, coronary sclerosis, or pulmonary emphysema influence the risk of operation? Statistical abstractions would have little place in helping the average patient make up his mind in favor of surgical operation. The experienced surgeon is well aware

of the importance of having his patient come to operation with mental composure, a factor which in itself may affect the outcome.

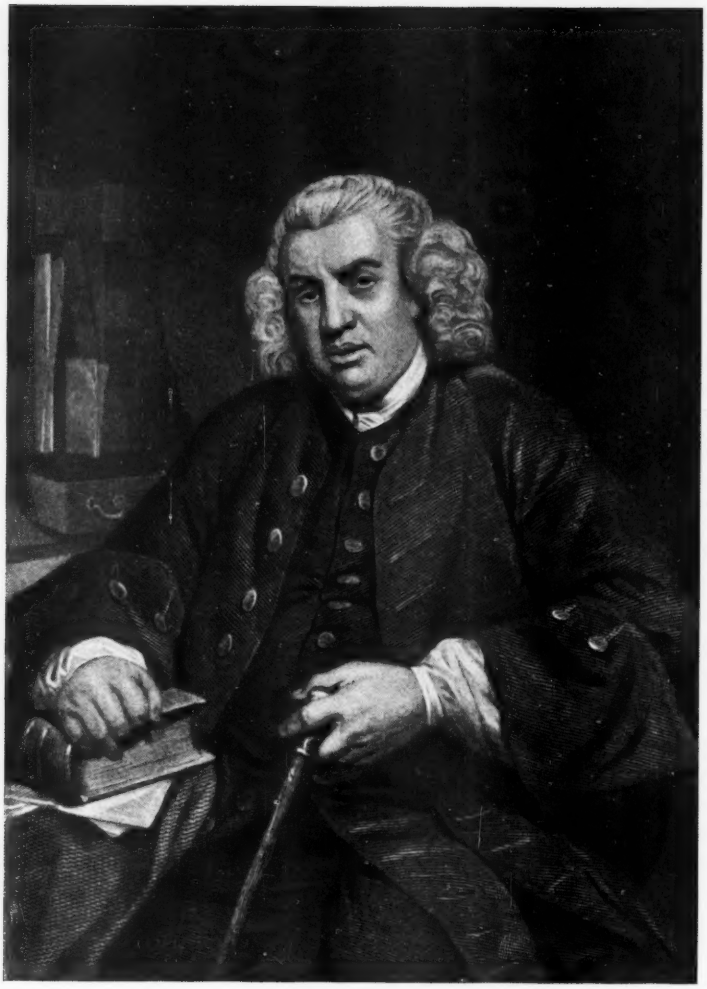
Assessment of the risk of operation is complicated by the continuous forward motion of surgery, and what may have seemed a reasonable hazard ten years ago may no longer be defensible. In the space of a few years after the discovery of vitamin K the mortality rate for operations on the bile ducts of jaundiced patients fell to one-half or less. Sulfonamides, penicillin, and streptomycin have changed the picture overnight and broadened the scope of surgery. Improvements in anesthesia and the knowledge of how to treat depleted or shocked patients have lessened the risk of operation and require constant re-evaluation of the question of surgical risk. Development of the residency training system reduces operative hazards and postoperative morbidity. Thus the surgeon is called on to estimate not a fixed value but a factor which is changing with general improvements in surgery as well as with his own increasing skill and experience. It is the obligation of referring physicians to keep these matters in mind also. It would be inexcusable for gastroenterologists, for example, to weigh the indications for colectomy in chronic ulcerative colitis in terms of the operative risk of twenty years ago.

The problem has special applications in relation to the uncommon surgical lesion, the new operation, or the specially complicated procedure. There are those who urge that the operative repair of strictures of the common duct should be done in a few centers by surgeons with unusual experience in the field. The operations for correction of vascular or of cardiac anomalies, or the shunt operations for portal hypertension, or resection of duodenum and pancreas tend to be practiced in only a few clinics, and unquestionably the results are better in a few rather than many hands. Should all patients who need such specialized surgery be referred to certain centers? The question is not easy, for there are personal, social and economic factors involved, as well as problems of graduate education and medical research. Nevertheless, for the surgeon with less experience who is called upon to perform such procedures the question has ethical and moral aspects, for he may sincerely feel that the patient is asking to be subjected to a greater risk than might result in a center for such surgery.

Since the risk of any major surgical operation will depend, among other things, on the condition of the patient the surgeon can influence the results in a series of cases by the extent to which he screens his patients and rejects poor risk cases. A too cautious attitude and anxious regard for mortality statistics on the part of the surgeon are not wholly commendable, for presumably he should be bent on doing the most good for the most patients. Nevertheless, it is possible to discredit an operative procedure by applying it as a last resort measure in desperate cases. Referring physicians as well as the laity in the community may lose interest, with the result that entirely suitable cases are not given their chance. This point is of special importance in smaller communities and in hospitals for chronic disease, notably in hospitals for tuberculosis. Furthermore, the surgeon himself may become disheartened and abandon the procedure. And yet, if a patient has one chance in ten of surviving an operation which promises cure and no chance of surviving without it, should he not be operated on? In bald form the question may seem easy, but the problem in practice is never so clear-cut.

Here is but one illustration of the intellectual challenge of surgery and of the comprehensive demands on the surgeon's personal qualifications.

JOHN D. STEWART



Samuel Johnson

1709–1784

From a painting by Sir Joshua Reynolds

THE SURGEON'S LIBRARY

THE BOOK SHELF

THE CASE OF THE ECCENTRIC LEXICOGRAPHER

ESTHER H. VINCENT, Evanston, Illinois

SAMUEL JOHNSON, so famous for his intellectual abilities, is almost equally famous for his physical disabilities. Indeed it was something of a shock to look upon him for the first time. Huge and grotesque, his tall, stooped figure was topped by a massive head with heavy, scarred features, the thick-lipped mouth keeping up a continuous business of opening and shutting. His great body was never quiet—the fingers twirling, the hands twisting, the feet shuffling, the trunk rolling about. His clothes, thrown on anyhow, presented a shabby medley of ill-fitting wig, wrinkled coat, soiled shirt, falling stockings and scuffed shoes. And yet, except for Shakespeare, no writer of English has a larger or more eager assembly of scholarly fans.

When Sam was born in Lichfield in 1709, his father, Michael, was already past fifty-two, and his mother, Sarah, was past forty. They had been married for some three years, and Samuel was the first of their two children. Besides keeping a bookshop at Lichfield, industrious Michael sometimes sold books in a stall at Birmingham, sometimes at other nearby towns. Michael wasn't much of a business-man, and, although he achieved a few local honors, he and Sarah were almost always hearing the wolf at the door. Sarah was inclined to be a bit arrogant about her family being better than Michael's, but the only member known to us is her nephew, the notorious "Parson Ford," said to be the clergyman sitting next to the punch bowl in Hogarth's "Modern Midnight Conversation."

Michael had a large, robust body and a strong, active mind, both of which he handed down to Samuel. But along with this goodly heritage went a "vile melancholy" that plagued the both of them all their days. Sarah seems to have been a woman of unique understanding and of deep religious feeling. Both Michael and Sarah lived to a good old age, Sarah reaching the age of ninety.

From the Archibald Church Library, Northwestern University Medical School.

The infant Samuel was far from promising. Born almost dead, he was unable so much as to cry out for a considerable time. But the man midwife declared: "Here is a brave boy!" Dr. Samuel Swinfen, the Johnson's young lodger, and one of Sam's godfathers, said that he never knew of any child so difficult to rear. The baby stayed for some ten weeks at the house of a wet nurse, who was blamed for his sore eyes. The eye trouble was probably tuberculous keratitis, but of course Michael and Sarah couldn't have been expected to realize it. A few weeks after birth, Sam developed some sort of lesion on his body, also, and while he was still nursing, an "issue" was cut in his left arm and left open until he was six. Sometime in his second year he was taken to a neighboring town to be examined by an oculist, and the next year to London. There, on the advice of Sir John Floyer, the distinguished physician, young Sam was "touched" for scrofula by Queen Anne, whom the child ever after remembered fearfully as "a lady in diamonds and a long black hood."

The little fellow was sent to a dame school and then to grammar schools at Lichfield and Stourbridge. Foolishly proud of him, Michael continually tried to make him "show off," thus causing the intelligent lad no end of humiliation. His phenomenal memory was considered a fair field for exploitation by his teachers, also. Even his school-fellows recognized the clumsy, diseased boy's intellectual superiority, and three of them used to mount him on their shoulders and carry him to school.

Two years were spent in "lounging," in binding, selling and reading books, and in making friends with Gilbert Walmesley and David Garrick. In 1728, young Johnson went up to Oxford, probably as result of a legacy left to his mother at that time. He stayed at the University for little more than a year, leaving for two reasons—too little money and too much melancholy. Hitherto he had compensated for poverty, disfigurement,and inability at sports by means of his intellect and a

certain gay wit that he was accustomed to use with much gusto. But now there came a sudden change. While on a short visit home, Johnson evidently suffered some mental crisis or accumulation of crises, and thereafter suffered eternal sadness. Oddly enough his gaiety kept company with his melancholy, making him a paradox, a pessimist with an enormous zest for life.

His mental depression drove him to seek relief from Dr. Swinfen, then of Birmingham, who so admired Johnson's self-written "case-report" that, forgetful of professional ethics, he showed it to several friends. According to Boswell, Johnson "felt himself overwhelmed with a horrible hypochondria, with perpetual irritation, fretfulness, and impatience; and with a dejection, gloom, and despair which made existence a misery." He attributed to paternal inheritance the melancholy which made him "mad all his life, at least not sober." It was the indiscreet Dr. Swinfen who suggested to the poor youth that his melancholy might some day lead to madness. But in spite of his fear of insanity and death, the courageous Sam yet maintained his gorgeous appetite for learning, literature, good food and good company.

On leaving Oxford, Johnson got a series of teaching jobs, none of them very satisfactory, and made his start in the literary field by writing essays, prefaces, and the like. In 1731 his father died, leaving his ailing business to Sarah and their younger son, Nathaniel. About this time too, Samuel met his future wife, Tetty Porter, a widow twenty years his senior, and the mother of three children. Their mutual attraction is still a mystery, but marry they did, in 1735, and lived happily together until Tetty's death in 1752, just as Johnson was beginning to be famous.

Meanwhile, Johnson, handicapped by an unprepossessing appearance, an indolent melancholy, poor eyesight and poor hearing, tried to set up a school of his own near Lichfield. This school lasted less than two years, but one of the few pupils was the dramatic David Garrick. Soon after, brother Nathaniel died, possibly by his own hand, and Tetty's daughter eventually went to live with the old mother.

Samuel was drawn inevitably to the big city, where he rambled along on his literary way, writing, grubbing, planning, talking and living hugely amid the elegant squalor of Hanoverian London. At first sight strangers sometimes mistook him for an idiot, as did Hogarth, who first saw him at the house of novelist Samuel Richardson, "standing at a window in the room, shaking his head, and rolling himself about in a ridiculous manner." Hogarth was struck with amazement when Johnson began to talk. Talking was necessary to Johnson, and in order to assure himself of a permanent audience, he founded a social club which later became the famous Ivy Lane Club. His list of friends grew long and interestingly varied—Garrick, Reynolds, Goldsmith, Edmund Burke and the gay Topham Beauclerk. Among the ten charter members of the Club were three physicians, William McGhie, almost the only Scotsman that Johnson ever could endure, young Edmund Barker, and "Dear Bathurst," the "good hater" whom Johnson loved.

In that day medicine existed for the benefit of its practitioners. In ordinary cases the physician did not bother to make a personal call. He used an intermediary, an apothecary, who reported the symptoms and received the Latin-written "bill" which specified the treatment. The apothecary then administered to the patient the clysters, purges, or emetics, and only called a barber-surgeon or a cupper when bleeding or cupping were necessary. The physician himself came only in serious cases and then charged a guinea or two-thirds of a guinea per mile. Consequently, the poor had to depend on unlicensed practitioners and quacks. Many people dosed themselves according to their own ideas. Johnson was not alone in his faith in "Dr. James's Powder for Fevers and Other Inflammatory Distempers," a dangerous mixture of antimony and phosphate of lime, concocted by an old home-town friend.

Johnson loved the company of medical men and often said that "they did more good to mankind without a prospect of reward than any profession of men whatever." He knew William Heberden, whom he regarded as "ultimus Romanorum," the last of the learned physicians. Then there was Dr. Nugent, Burke's father-in-law, and Dr. Fordyce, a St. Thomas's staff member, a great gourmand, and the author of a popular textbook of medicine. When Johnson thought himself seriously ill, he sometimes consulted his old friend, Dr. Thomas Lawrence, a past-president of the Royal College of Physicians. Lawrence and Johnson had a fellow-feeling for one another, as both were the victims of strange tics and convulsions, and both were hard of hearing. Usually, however, Johnson sought the advice of a member of his own household, Robert Levett.

Levett was one of those queer dependents whom Johnson gathered around him, partly to serve as an outlet for his natural compassion, partly to keep him company after the death of Tetty. Levett and poor, blind Anna Williams, together with the negro servant, Francis Barber, were the permanent occupants of that nest of odd birds

that kindly Samuel kept in his house. Levett was an uncouth fellow, a forlorn, middle-aged hanger-on, who had been apprenticed to a Hull draper, had gleaned some medical lore from a neighbor, had drudged in London to make money enough to take him to Paris, where he worked as a waiter in cafés frequented by surgeons. For five years he got free tickets to the best French lectures on the medical sciences. Then he returned to London where he healed the poor for a crust of bread, a glass of gin, a few coins, or whatever they had to give him. He continued to attend such lectures as were free, and may be said to have gained his medical education by ear.

Johnson had very definite ideas on the subject of medicine, and his views were considerably ahead of most of the physicians of his time. He disapproved of periodical bleeding, and also of the current eighteenth century practice of polypharmacy. He fancied himself as a writer of prescriptions, not only for his own ills, but for those of his friends. To rheumatic Bennett Langton he sent a formula for sulphur, mustard seed, sugar and infusion of the root of lovage. To Dr. Lawrence, who suffered hemiplegia and aphasia, he suggested electrical treatments, and to Miss Boothby he prescribed powdered dry orange peel for dyspepsia.

He seems never to have paid fees, once remarking, "Illness is of little expense to me, thanks to the generosity of my physicians." He may have given them books in exchange for treatment, for when Dr. Warren attended him in his last illness, he said, "Sir, you come at the eleventh hour, but you shall be paid the same as your fellow-labourers. Francis, put into Dr. Warren's coach a copy of the *Lives*." As a patient he was most trying, constantly interfering with his treatment. Long-suffering Dr. Lawrence once cried, "If he would come and beat me once a week, I could bear it. But to hear his complaints is more than I can support." Johnson kept a Latin diary of the state of his health and of the effect upon it of the remedies used.

Never a worshipper of Bacchus, Johnson eventually gave up alcoholic drinks altogether, and substituted tea-drinking to the extent of fifteen or sixteen cups a day. During his later years, tea, talk and lemonade were his only solace. Smoking he never cared for.

The world has reason to be grateful to George III, whose well-intentioned stupidity finally blundered into giving the Great Moralist a pension. Within five years of gaining economic security, Samuel had moved into a comfortable house, had met Boswell, had begun his happy friendship with the Thrales, had founded, with Sir Joshua Reynolds, the famous Club, and had begun the series of visits and ramblings that reached their climax in the tour to the Hebrides. He had now become a public personage, often satirized and caricatured, but nevertheless a sort of British institution. At his morning receptions he drank tea in bed and declaimed between sips. At noon he would dine in a tavern, stay until late afternoon, drink more tea at the house of a friend, and eventually retire supperless to his home to read and write far into the night.

Dr. John Lettsom says of him at this period, "In company I neither found him austere nor dogmatical; he certainly was not polite, but he was not rude; . . . he was sometimes jocular, but you felt as if you were playing with a lion's paw. His body was large, his features strong, his face scarred and furrowed with scrophula; he had a heavy look; but when he spoke it was like lightning out of a dark cloud."

His last days were embittered by the death of Henry Thrale and by the defection of Henry's widow, Hester. Apoplexy claimed the food-loving Henry, and Mrs. Thrale, probably Johnson's dearest friend, instead of centering her life around Johnson, as he had expected, became infatuated with a young Italian singer named Pozzi, and was lost to Johnson as well as to English society.

Johnson was a robust man, but not a healthy one. He never knew a day's complete ease in his life. But in spite of his ailments, he had great muscular strength, was a big eater, and was capable of sitting up to all hours. When he was sixty he bathed in the sea at Brighton in October. At sixty-five he had an attack of gout, and at sixty-seven he had another attack, together with "difficult and laborous respiration" for which he was bled rather heavily. The next year he said, "The hand of time or disease is heavy upon me. I pass restless and uneasy nights, harassed with convulsions of my breast, and flatulencies of my stomach."

At the age of forty-six he had had laryngitis, but no further respiratory diseases until he was sixty-three, when he was plagued by a winter cough which recurred at intervals for some years. Venesection was the treatment. Asthma laid him low during the winter of 1781–82, and for the rest of his life he breathed with difficulty. Opium brought him great relief, and his dose rose to three grains daily. But on his seventy-second birthday, he thought he had more bodily strength and vigor than was common.

Dropsy was first noticed in 1783 by Sir Lucas Pepys. Diagnosis of sarcocele was made by Per-

civall Pott in consultation with William Cruikshank, and it was put up to Cruikshank to make an excision. But Cruikshank was afraid of operative shock, and while he delayed, "the sarcocele ran off at an orifice made for mere experiment." At seventy-four Johnson had a stroke. He wrote to Mrs. Thrale:

"I went to bed, and in a short time waked and sat up as has long been my custom, when I felt a confusion and indistinctness in my head, which lasted, I suppose, about half a minute. I was alarmed, and prayed God that however He might affect my body, He would spare my understanding. This prayer, that I might try the integrity of my faculties, I made in Latin verse. The lines were not very good, but I knew them not to be very good; I made them easily, and concluded myself to be unimpaired in my faculties. Soon after I perceived that I had suffered a paralytic stroke, and my speech was taken from me."

He tried to treat himself with wine, thinking "to rouse the vocal organs." Surprised at his own apathy, he sent for Brocklesby and Heberden, who blistered him and gave him salts of hartshorn. Recovery from the hemiplegia and aphasia was rapid and complete.

Later in the year gout plagued him again, and he had an attack of spasmodic asthma of great violence that forced him to sit up all night. Dropsy became grave, increasing to the thighs, and by February of 1784 was so extensive that Johnson had Boswell consult five Edinburgh physicians, including Cullen, Hope and Monro. The five, despite the disadvantage of long-distance prescribing, recommended squills, which Johnson took with such vigor as to produce unpleasant reactions. His only other remedy was rhubarb, then a rare and costly drug. He was aware now of precordial constriction on exposure to cold, and had to take tincture of laudanum for night dyspnea. "My diseases are an asthma, and a dropsy, and what is less curable, seventy-five," he said.

About the middle of March, after a day of prayer and fasting, Johnson had a urinary crisis, passed twenty pints of urine, and became free from the dropsy that had kept him indoors for over a hundred days. After a trip to Oxford and Lichfield which tired still further his already tired heart, the dropsy returned, but was controlled by drugs and rest until November. In December the edema was so great that his legs were incised. To poor Cruikshank he cried, "Deeper, deeper; I will abide the consequence. You are afraid of your reputation but that is nothing to me!" Still not satisfied that the incisions were deep enough, he got a lancet and cut himself again and then thrust a pair of scissors into the calf of each leg. Because of this action a rumor started that he had committed suicide.

When Dr. Warren expressed the hope that he was better, the death-fearing Johnson bravely replied, "No, sir, you cannot conceive with what acceleration I advance towards death!" And when Dr. Brocklesby told him that there was no hope for him, he said, "I will take no more physic, not even my opiates; for I have prayed that I may render up my soul to God unclouded." And so he died.

A post-mortem examination was performed in the Library of the Royal College of Physicians two days after death by Dr. James Wilson, Cruikshank and Heberden being present. The lungs did not collapse but remained distended, apparently without the power of contraction. The air cells on the lung surface were much enlarged, and the right lobe adhered strongly to the diaphragm. No water was found in the thorax cavity. The heart was very large and the valves of the aorta were beginning to ossify. Not more than the usual amount of fluid was in the pericardium. Incipient peritoneal inflammation and ascites appeared in the peritoneum. The liver and spleen were large and firm, the spleen having almost the feel of cartilage. The gall bladder contained a stone the size of a pigeon's egg. The account also mentions "hydatids" in the right kidney and destruction of the left kidney with two very large hydatids formed in its place. These were probably common renal cysts, not distinguished from hydatids until 1793, when Matthew Baillie published his book on morbid anatomy.

The story is that of a man with good family history, who acquired gout and a hypertension leading to a transient stroke. Emphysema aggravated the symptoms of heart failure. Rest, squills and opium brought some relief, but too much travel brought on another attack, and death ensued.

Like many literary men, Johnson suffered much from impaired vision. Fanny Burney spoke of him as looking at the books in her father's library "almost touching the backs of them with his eyelashes as he read their titles." His short sight would explain his lack of interest in pictures, sculpture and architecture, just as his defective hearing would explain his lack of interest in music. According to Treves his poor vision was due to a leucomatous change in the corneae as a result of tuberculous keratitis, probably connected with the tuberculous glands in his neck. In spite of treatment, the sight of the left eye was lost. Johnson was very sensitive about his myopia and never wore spectacles. Twice in his life his right eye became inflamed from much reading of small print.

His gastrointestinal symptoms were probably the result of his erratic eating. His gallstone may have been pure cholesterol, but the adhesions found between the folds of the jejunum could have been caused by cholecystitis.

Some endocrine disorder may have produced his bulky form and massive features. Probably there was some degree of hyperpituitarism combined with some hypothyroidism. His hands were not unduly large.

A strong sense of inferiority could very well result from a childhood such as Johnson's. Scrofula and defective vision combined with a clumsy body kept him from mixing with other boys at games. His ego instincts could find outlet only in intellectual distinction. He no doubt found partial compensation, too, in fantasy and in the friendship of young, handsome, gay men, like Beauclerk, for instance. His melancholy and his tics were marks of his frustrations. His melancholy he tried to mitigate by constant companionship—he had a morbid dread of being alone. His tics were only habit spasms or motor tics. As he walked along a street, he felt obsessed to touch certain posts, turning back if he missed one.

The suggestion has been made that Johnson was sexually impotent. To be sure, he married, but his bride was an elderly widow, and he may have felt that he could marry her without doing her any injustice. Boswell says that he was invariably chaste, but also that his amorous side was very strong and that he used to take women to taverns in order to hear them relate their stories. This could have been an indirect method of sexual satisfaction. And his declared wish not to have children could have been a rationalization of his knowledge that he never could have any. Be that as it may, he certainly suffered from some inner emotional conflict which tormented him all his days.

He was buried in Westminster Abbey among the great of Britain, "a true-born Englishman of the eighteenth century."

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REVIEWS OF NEW BOOKS

THE book, *British Surgical Practice*,[1] is the third in a series of volumes, two of which have been reviewed previously. The subjects, presented alphabetically, extend from "Cesarean Section" through "Eyelids."

Once again there is a profusion of illustrations throughout the book which are pleasing in their simplicity and add a great deal to the clarity of the work. The chapter on cesarean section is well outlined. In fact, the entire book continues what is hoped will be the practice throughout all the volumes; namely, that of supplying not only clear chapter and subchapter titles but also convenient marginal index labels. Perhaps the most significant part of the chapter on cesarean section is the full list of indications. In any discussion of this topic the indications come in for close scrutiny, and apparently in all cases surgeons are subject to criticism should promiscuity be the practice. The usual varieties of the operation are outlined in a simple, clear fashion, and the illustrations are well done.

It is interesting to note that an entire chapter is devoted to tumors of the carotid body. Recently in the United States, authorities have been outspoken in their frank admission of theories which have heretofore been held rather secret. Lahey is now emphasizing repeatedly, and his lead is being followed by other authorities, that when such a tumor involves the great vessels in such a way that a sacrifice of the vessels will result in not only a high incidence of hemiplegia but also a forbidding mortality rate, such treatment is decidedly out of order. The author of this chapter agrees with the middle group of American surgeons who feel that only 20 per cent of such tumors are malignant in the first place. One will recall the argument used in this country today by some outstanding authorities that there is a possibility of malignancy in 50 per cent. The diagnosis of carotid body tumor is made quite evident, and the treatment suggested agrees very closely with that accepted at present in this country; namely, that if exploration reveals too great a risk in considering removal, the surgeon should be satisfied with biopsy alone.

There is some general discussion on cellulitis, lymphangitis, erysipelas and so forth. This discussion is divided into two parts; namely, that on general infections and that on localizing infections, such as those in the pelvis. Chemotherapy is mentioned, but there is a notable lack of discussion in regard to penicillin and streptomycin. One derives the impression that the chapter was written prior to the general availability of the latter agents, although in the discussion of the cases of pelvic cellulitis it appears that penicillin has indeed come in for its logical usage.

A chapter on the cervical rib and the scalenus syndrome is complete and offers a good bibliography, especially referring to the work of Adson.

Perhaps a bit out of place in this volume, from the viewpoint of the casual American surgeon, is the inclusion of a discussion of chemical warfare and its surgical aspects. Probably those persons who were not subject to the mass bombings would not be as apprehensive about the possibilities, but one can appreciate the thoughts that must have gone through the mind of the British surgeon at that time. The discussion is timely and agrees well with the material promulgated during the recent war by not only the American army and navy medical departments, but also the civilian defense groups.

There is a separate chapter on chemotherapy which is delightful; the average volume on surgery would overlook such a handy outline. The selection of the various drugs is discussed, their methods of administration are rendered crystal clear, their toxic effects are pointed out and the beneficial results to be expected from the chemotherapeutic agents are discussed with frankness.

There is a section on coagulants and anticoagulants. The preliminary outline of the mechanisms of clotting and bleeding, as well as a discussion of the prothrombin factors, is very timely. The various agents for coagulation are mentioned briefly, with a short characterization of each. The reader is not told, but one gets the impression, that perhaps in Great Britain these agents are employed more widely than they are at present in the United States. The anticoagulants, namely, heparin and dicumarol, are given proper space. Fortunately, there is a good list of references here which those seriously interested would do well to employ.

In the chapter on colitis the outstanding feature is the illustrations regarding various procedures to be used occasionally. Several types of cecostomy and appendicostomy are stressed. Recently it has been stated in the United States by many authorities that "open operations" upon the colon itself have little place in cases of colitis. It is felt that since the disease is usually so generalized in the colon, the correct part of the intestine to be employed is the ileum. This is not made clear in the present volume. Ileostomy, which is employed extensively in the United States, comes in for a rather thorough discussion later in the chapter. The illustrations once again are very good and the technique is given thorough discussion. Fortunately, there is also a discussion of the appliances that may be used, together with

[1] BRITISH SURGICAL PRACTICE. Under the General Editorship of Sir Ernest Rock Carling, F.R.C.S., F.R.C.P., and J. Paterson Ross, M.S., F.R.C.S. Volume 3. London: Butterworth & Co., Ltd.; St. Louis: The C. V. Mosby Co. 1948.

agents for protecting the skin. Apparently, the British surgeons experience the same types of complication that we experience in this country; namely, prolapse of the ileum, formation of abscesses and fistulas, and intestinal obstruction. The writer is well advised in generalizing his statements in regard to colectomy in this disease. The exact time interval between ileostomy and colostomy remains controversial, although there is more and more tendency now toward uniformity. The possibility of carcinoma arising in the diseased colon is mentioned along with other complications, which should remind the surgeon to resort to colectomy at the optimal time.

There is a rather long and complete chapter on carcinoma of the colon by no less an authority than Sir Hugh Devine. This offers almost all that the average practitioner needs to know about the field today. In addition to the thorough discussion of the clinical features of the disease, the various methods of dealing surgically with carcinomas located at different points of the colon are outlined. It is difficult to guess just when this chapter was written. The role of chemotherapy is given full credit and the failure of penicillin to be effective against the Bacillus coli is stressed. However, the operative techniques seem rather old fashioned and, in fact, in many centers in the United States today would be considered so. The so called extraperitoneal resection of the terminal part of the ileum and right portion of the colon leaving a double barreled stoma is now largely reserved for emergency use in this country. Actually, the wide acceptance of one stage resection with primary anastomosis has been one of the most gratifying advances in this field of surgery. Devine admits that the modern adjuncts to preoperative care may indeed change the picture somewhat, but he expresses the opinion that these adjuncts are to be used to render his already safe operation even safer. His method of immediate anastomosis performed in low lying lesions of the colon always requires previous defunctionalization of the colon with its attendant complications, prolonged morbidity and, a factor which can be overlooked easily in the final analysis, a mortality rate which must be computed on a many stage operation. Even today the proponents of the multiple stage operation will quote a mortality rate which represents the resection only. All clinics are aware of the accidents, admittedly uncommon, which attend crushing the colostomy spur and, in addition to this, the leaks which can be observed after an intraperitoneal closure of the colonic stoma. The controversy probably still rages but no one can deny that in the leading centers throughout the United States today, qualified surgeons are so pleased with primary resection and immediate anastomosis (without the use of a "vent" or "safety valve") performed in the selected case of the properly prepared patient that they are certain they will never have to return to the more obsolete method. Devine omits discussion of carcinoma high in the rectum, but his views are so completely covered in his other articles to which he refers in this chapter that the careful student will become quite familiar with it.

In a chapter on megacolon the treatment presented concerns itself entirely with sympathectomy. Certain authorities in this country, notably Dixon, are now teaching that resection of the involved intestine wherever possible will give a more satisfactory result.

There are many chapters dealing with items that can scarcely be of interest to the average surgeon, which will serve as a handy reference in an uncommon instance. These include conjunctivitis, eye injuries, operations about the ear and labyrinth, and so forth.

The chapter on diverticula of the alimentary tract is good in that it points out that almost the entire tract may be involved.

There is an interesting section on endometriosis which embraces several of the leading theories on the cause of this condition. The clinical picture is made rather clear cut and the treatment agrees with that taught in the United States. Of special importance is the author's advice to leave intestinal lesions alone unless there is actually acute obstruction, since the intestinal lesions will regress after oophorectomy. The young surgeon will be well advised to leave alone those lesions which are too extensive to remove without damage to vital structures since termination of the ovarian function will control the disease. In the author's words, "There is no justification for attempting to eradicate the disease if the operation is likely to endanger the patient's life." The list of references for this topic is disappointingly short.

There are some chapters devoted to aids to diagnosis which include peroral endoscopy as well as cystoscopy and sigmoidoscopy. The trend in the United States today is toward collaboration in diagnosis, since every field is now so complicated and intricate that it is difficult for any one man to master all of the modern ramifications of all fields. Therefore, a busy surgeon may do well to seek diagnostic help from a qualified colleague when it comes to the expert manipulation of an instrument which must be applied in what is admittedly a blind procedure. The world literature is notably lacking, but the memories of the older surgeons are replete when it comes to recalling distressing and even fatal accidents which have occurred in the injudicious use of these blind procedures.

E. S. JUDD, JR.

THE third edition of *Control of Pain in Childbirth*[1] is considerably enlarged and improved. There are now 522 pages as contrasted with 356 pages in the first edition. Special attention is paid to the technique of caudal and continuous caudal analgesia, spinal and continuous spinal analgesia, and the saddle block spinal analgesia. There is also a discussion of the use of demerol during labor.

[1] CONTROL OF PAIN IN CHILDBIRTH. By Clifford B. Lull, M D., F.A.C.S., F.I.C.S., and Robert G. Hingson, M.D., F.I.C.S., F.A.C.A., F.I.C.A., with an introduction by Norris W. Vaux, M.D. 3d ed. Philadelphia: J. B. Lippincott Co., 1948.

The arrangement of the chapters is essentially the same as it has been. The last 25 pages of the text are devoted to an interesting article on "An Analysis of Newborn Mortality and Morbidity, With Controls in New York, Philadelphia, and Memphis with Continuous Caudal Anesthesia" which was written by Hingson, Edwards, Lull, Whitacre, and Franklin. Many new illustrations and charts have been added.

This book is useful because it contains a huge amount of valuable data which are analyzed in an unbiased manner. Likewise, there are included in the book several sections prepared by collaborators who are experts in special methods of administering pain relief to women in labor. This book is highly recommended to all who are interested in obstetrics and anesthesia. J. P. GREENHILL.

CORRESPONDENCE

SUBMUCOUS LIPOMA OF THE COLON.—A Correction

A LETTER from Dr. Charles W. Mayo advises that an error was made in the manuscript for the article entitled "Submucous Lipoma of the Colon" by himself and Dr. Donald F. Griess, in the March, 1949, issue of SURGERY, GYNECOLOGY AND OBSTETRICS. Paragraph 3, page 309 should read as follows:

"Comfort reported 28 cases of submucous lipoma of the gastrointestinal tract, most of which were found at necropsy. However, there were 3 cases in which operation had been performed prior to 1928. In these 3 cases, 1 lipoma was situated in the sigmoid, 1 in the hepatic flexure and 1 in the ascending colon. Pemberton and McCormack reported 3 additional cases of submucous lipoma in which operation had been performed between 1928 and 1936. In this series, 1 of the lipomas was situated in the hepatic flexure, 1 in the descending colon and 1 in the ascending colon. The 19 cases which we are reporting in this paper make a total of 25 in which operation was performed at the Mayo Clinic up to and through the year 1947. Some of the cases in this total series have been reported individually, previously."

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgement must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

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